1-1-1967

Effects of chlordiazepoxide on the amygdala and hippocampus of conflicted squirrel monkeys.

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GREEN, Kenneth Fisher, 1940-

EFFECTS OF CHLORDIAZEPOXIDE ON THE AMYGDALA AND HIPPOCAMPUS OF CONFLICTED SQUIRREL MONKEYS.

University of Massachusetts, Ph.D., 1967
Psychology, experimental

EFFECTS OF CHLORDIAZEPOXIDE ON THE AMYGDALA AND HIPPOCAMPUS OF CONFLICTED SQUIRREL MONKEYS

A Dissertation Presented

by

Kenneth Fisher Green

Submitted to the Graduate School of the

University of Massachusetts,

Amherst, in

Partial Fulfillment of the Requirements for the Degree

of

DOCTOR OF PHILOSOPHY

January, 1967

Major Subject: Psychology

EFFECTS OF CHLORDIAZEPOXIDE ON THE AMYGDALA AND HIPPOCAMPUS OF CONFLICTED SQUIRREL MONKEYS

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January 1967
(Month) (Year)

Acknowledgement

I wish to thank Drs. Robert S. Feldman, Chairman, Jay A Trowill, and Theodore D. Sargent for serving on my Guidance Committe. The decisions and suggestions of these members improved this Dissertation. The comments of Drs. John W. Moore and Lawrence Frase, who served as readers, are also gratefully appreciated.

Partial support for this project was furnished by United States Public Health Service Grant M-01061 to Dr. Robert S. Feldman and by the Psychology Department.

I also wish to thank Drs. John W. Moore, Seymour Epstein, and Stuart P. Taylor for the loan of recording equipment, and Messrs. Jay Isgur, Joseph Mach, and Allen Williams and Mrs. Nancy Feldman for technical assistance and advice.

A special note of thanks is due my wife, Carolyn, who sympathetically tolerated the many changes of mood associated with the performance and completion of this project.

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Introduction

It was the purpose of the present research to investigate the electrical activity of the amygdala and hippocampus while squirrel monkeys were adopting and maintaining a stereotyped response in the Maier paradigm. It was further intended to investigate the effects of chlordiazepoxide, a tranquilizing drug which has been shown to prevent stereotyped responses from developing into fixations, on the activity of these structures and on the behavior.

The Maier paradigm (Maier, 1949) is a two-stage procedure in which animals responding on a two-choice discrimination problem are rewarded for "correct" responses and are punished for "incorrect" responses. In the first stage there is no consistently correct cue and animals typically adopt a stereotyped response, i. e., none of the cues of Bright, Dark, Left, and Right is consistently rewarded and animals typically respond exclusively to one cue. In the second stage a cue other than the one to which the response is directed is designated as correct, but the animals seldom depart from their stereotypes in order to achieve the more favorable adjustment. Animals do, however, show by differential latencies that they "know" the solution to the soluble problem. If, for example, they are stereotyped to a position and one of

the nonspatial stimuli is correct, they will respond sooner to the correct cue than to the incorrect cue but will not deviate from their stereotyped responses. The consistency with which the insoluble-problem contingencies produce stereotypes and the stability of the differential latencies in the soluble problem have suggested that the Maier paradigm might furnish valuable baselines for the evaluation of psychotropic drugs.

Feldman (1964a; Feldman & Lewis, 1962) reviewed Maier paradigm experiments in which rats responding to a Dark-Bright discrimination on a Lashley jumping stand were given tranquilizing or energizing drugs in the insoluble-problem phase, in the soluble-problem phase, or in both phases. No drug given only during the soluble-problem phase of the Maier paradigm had significant effects in preventing or alleviating fixations.[1] However, chlordiazepoxide (CDP) and diazepam (DZP), both members of the benzodiazepine class of tranquilizers, were found to have potent fixation-preventive properties if given only during the insoluble problem. In the subsequent soluble problem, 73% of the CDP rats solved (Feldman, 1962) and 65% of the DZP rats solved (Feldman, 1964b). In addition, CDP given during both phases allowed 40% of the rats to solve (Feldman, 1962). No other drug produced such dramatic results,

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1. An animal is said to be fixated if it practices its stereotyped response for a criterional number of trials in the soluble problem.

although one energizing drug (phenelzine) given only during the insoluble problem yielded 40% solutions (Bremner, 1960). CDP, DZP, and phenelzine effected a decrease in latencies over days in the insoluble problem, and effected immediate decreases in latencies when given to fixated Ss in the soluble problem. On the other hand, drugs which lacked fixation-preventive properties were found to increase latencies in both phases of the Maier paradigm. Thus, two classes of drugs may be identified according to their effects on behavior in the Maier paradigm: (1) those which have been shown to prevent fixations and to reduce latencies and (2) those which have been shown not to prevent fixations and to increase latencies. The remainder of this discussion will be limited to CDP, which is perhaps the best-known benzodiazepine and, as described above, is the most potent of the fixation-preventing, latency-reducing drugs.

Tranquilizing drugs apparently achieve their effect of calming without sedating by acting upon rhinencephalic structures (McGeer, 1962). The structure within the rhinencephalon which is most susceptible to tranquilizers is the amygdala (Himwich, 1960), a structure which is importantly involved in modulating many types of emotional and motivated behavior (Gloor, 1960; Goddard, 1964). CDP has been shown to depress the amplitude and duration of evoked afterdischarges in the amygdala (Schalleck & Kuehn, 1960), to combat seizures induced by direct

electrical stimulation (Lanoir, Naquet, & Requin, 1964), to elevate the threshold for behavioral responses produced by amygdala stimulation, to decrease the amplitude of the evoked response in the hippocampus produced by amygdala stimulation (Morillo, Revzin, & Knauss, 1962), and to depress only slightly the spontaneous activity of the hippocampus (Schalleck, Zebransky, & Kuehn, 1964). These results suggest that CDP has more of an effect on the amygdala than it has on the hippocampus. Further support for this notion is offered by investigations of the evoked response of the hippocampus to electrical stimulation of the contralateral hippocampus. Such investigations conducted with CDP are unknown to the author, but DZP, which acts almost identically to CDP in the Maier paradigm (Feldman, 1962; 1964b) and in the rhinencephalon (Morillo, 1962a), was found by Morillo (1962a) to elevate the interhippocampal evoked response. This result suggests that CDP may suppress the mechanism described by Kriendler and Steriade (1964a; 1964b) by which the dorsal amygdala can suppress the hippocampal discharge produced by stimulation of the ventral amygdala in cats. In other words, CDP, by virtue of its depressive effect on the amygdala, may free the hippocampus of the amygdala's influence.

The hippocampus has recently been implicated in the ability of animals to learn new responses, or, more precisely, to suppress old responses. In simple approach

situations, hippocampal rats learned as rapidly as controls to run down an alley but extinguished more slowly than controls (Jarrard, Isaacson, & Wicklegren, 1964), and hippocampal cats acquired a panel push response as rapidly as controls but took longer to extinguish (Peretz, 1965). Passive avoidance has been found to be disrupted by hippocampectomy in rats (Kimble, 1963; Teitelbaum & Milner, 1963), indicating that hippocampal animals have difficulty giving up newly-punished responses as well as newly-nonrewarded responses. Hippocampal rats also have been shown to have difficulty in learning position-reversal shifts in T mazes (Thompson & Langer, 1963; Thompson, Langer, & Rich, 1965) and Y mazes (Lash, 1964; Kimble & Kimble, 1965). Therefore it may be suggested that some form of hippocampal disfunctioning might be induced in the Maier paradigm which might be responsible, in part at least, for the characteristic perseveration of stereotyped responses.

Although both the amygdala and hippocampus are parts of the brain's systems of structures for the control and release of behavior (Gerbrandt, 1964; 1965), the hippocampus is here viewed as being more likely than the amygdala to underlie perseveration in the Maier paradigm. For, while hippocampal animals tend to perseverate in earlier-learned responses (Ellen & Wilson, 1963), recent evidence shows that amygdalectomized animals will readily abandon a well-learned response either by inhibiting it (Horvath, 1963) or by exchanging it for an approach to a one different

from the original (Scwartzbaum & Poulos, 1965). Thus for the behavioral phenomenon in question, perseveration of a stereotyped response in the Maier paradigm, one can reasonably argue that the hippocampus is more immediately involved than the amygdala because hippocampectomy induces perseveration while amygdalectomy does not.

If the hippocampus is indeed more immediately involved than the amygdala in the persistence of stereotyped responses, then why should drugs which depress the amygdala prevent stereotypes from persisting to the point of becoming fixations? The answer can only be complex, and the present limited investigation can at best supply only a partial solution to the problem. The following hypothetical and possibly oversimplified interpretation may be offered.

During the insoluble problem phase of the Maier paradigm, high arousal may increase the activity of the amygdala to a high level. This notion is supported by work with cats in which a 40/sec. rhythm from the amygdala was found to increase as arousal increased (Gault & Leaton, 1963; Pagano & Gault, 1964) and in which the 40/sec. rhythm was found to be specifically elicitable by a stimulus associated with pain shock (Lesse, 1960). In addition, evidence which suggests that decreased amygdala activity is not consistent with fixations is offered by studies in which CDP and DZP have been shown to depress

the amygdala (Schalleck & Kuehn, 1960; Schalleck, Zebransky, & Kuehn, 1964; Morillo, 1962a) and to prevent fixations (Feldman, 1962; 1964b). Eventually the increased activity of the amygdala may produce a disfunction in the hippocampus, perhaps by acting through the transmission system described by Kriendler and Steriade (1964a; 1964b) and by Morillo (1962b). Since perseveration developed in the Maier paradigm does not transfer to other situations (Feldman, 1953), it is suggested that the disfunction in the hippocampus is situation-specific and might be viewed as a temporary lesion. The disruption of hippocampal functioning produced by extremely high amygdala activity may well persist into the soluble problem phase and, since the hippocampus is incapable of exercising its normal function, a stereotyped response would be allowed to become fixated. When an animal is given a benzodiazepine during the insoluble problem, amygdala activity is kept in check and the hippocampus is freed from at least the direct influence of the amygdala. Removal of the drug concurrently with the introduction of the soluble problem might thus allow the drugged animal to function more normally than a nondrugged animal in what is basically a simple learning situation.

The present research thus had two purposes. The primary one was to investigate the electrical activity of the amygdala and hippocampus during the insoluble problem

phase of the Maier paradigm when a stereotyped response was being adopted, and during the soluble-problem phase when the stereotype was persisting (becoming fixated) in the face of a more adaptive alternative. It was hoped that some aspects of amygdala and hippocampal activity could be correlated so that the proposed functional relation between the two structures might be confirmed. The second purpose was to establish that the hippocampus was involved in the inability of an S to give up a stereotyped response in the soluble problem. Comparison of hippocampal records during the insoluble problem and especially during the soluble problem of Ss given CDP in the insoluble problem to the like records of Ss given no drug was expected to shed light on this issue. If hippocampograms of CDP Ss differed significantly from those of nondrugged Ss, then the hippocampus would be strongly implicated in the establishment of fixations.

Method

Subjects.

The Ss were 10 young adult male and female squirrel monkeys (Saimiri sciureus) from the Psychology Department colony. Three (Bengt, Gunnar, and Ingmar) were experimentally naive, while the other seven (Anton, Dodie, Frieda, Gustav, Phaedra, Ulrich, and Winifred) had served in an experiment in which color preference was measured (Green, Moore, & Sargent, 1966) and one in which extensive color discrimination training was given (Green & Moore, 1966). During the experiment the weights of the Ss ranged from 500 to 1140 gm.

Surgery.

All Ss were given Bicillin (0.2-0.3 cc., i. m.), an antibiotic, on the day preceding surgery. One hour prior to anesthetization with Nembutal (22 mg./kg., i. p.) the Ss were tranquilized with Taractan (2.2 mg./kg., i. m.). Surgical conditions were as aseptic as practicable. Bipolar electrodes, which were directed bilaterally towards the amygdala and hippocampus, consisted of a pair of 0.014-in. diameter stainless steel wires twisted together and mounted in a socket as described by Valenstein, Hodos, and Stein (1961). The wires were insulated by an enamel coating except at the tips. The greatset distance across both wires at the exposed tips was 0.030 in. The coordinates used on the Kopf stereotaxic instrument, as

selected from the atlases of Gergen and MacLean (1962) and Emmers and Akert (1963), were 10.0 mm. anterior (from the interaural line), 7.5-8.0 mm. lateral (from the midline), and 18.0-21.0 mm. ventral (from the skull surface) for the basolateral amygdala, and 1.5-2.5 mm. anterior, 7.5-8.5 mm. lateral, and 17.5-20.0 mm. ventral for the posteroventral hippocampus. Small stainless steel screws were turned into the skull and were infiltrated with dental cement which was formed into a cap which held the electrodes. Prophylaxis against infection was effected by sprinkling sulfa crystals on edges of incisions.

Surgery occurred during the final phases of pretraining, and at least one week was allowed for recovery.

Apparatus.

The behavioral apparatus has been pictured elsewhere (Feldman & Green, 1966). The Ss were seated in a Foringer squirrel monkey chair and were restrained by plates at the waist and neck. In the neckplate were two handholes, one for the left hand and one for the right, which allowed S to reach half the display panel with each hand. The display panel was a sheet-metal rectangle (4.75 X 8.25 in.) containing two illuminable discs (Grason-Stadler Model E 8670 A) which were 1.25 in. in diameter and which served as both manipulanda and discriminanda. The display panel was mounted vertically in front of S and contained a delivery tray into which 75-mg. banana pellets (CIBA) were

delivered when a response was rewarded. The monkeys' tails were shaven on the underside, extended outside the back of the chair, and had a pair of shock electrodes (adapted from Stoelting finger electrodes, catalog number 24222) attached. Electrode paste was used to insure electrical contact. The restraining chair was enclosed in a sound-attenuating, electrically-shielded box which contained a sliding floor for easy installation and removal of Ss. Centered in the rear of the box were a pair of 24 V. DC houselights, mounted near the ceiling, and a bakelite panel holding connectors for electrode leads (Microdot cable), mounted 5-6 in. above S's head. A relay, which shorted the two leads from each brain electrode during tailshock administrations and thus prevented interaction between the two types of circuit, was also mounted on the bakelite panel. A second relay, which completed the tailshock circuit during shock administration and otherwise connected S to ground, was mounted behind S outside the box. The box contained a pair of observation windows, one on each side of S, and a loud fan for ventilation and masking of environmental noises.

Control and recording facilities were located in an adjacent room. Control facilities included a pair of programmers operated by punched tapes which determined which of the two discs would be illuminated on each trial. Timers, counters, relays, a pellet dispenser control, and

constant-current 60-cycle AC devices for delivering two levels of tailshock (0.5-1.0 ma. for goad and 4.0-4.5 ma. for punishment) were provided. A Lehigh Valley randomizer (Model 1485) was used to determine punished and rewarded cues during the insoluble problem phase of the experiment.

Electroencephalographic (EEG) recordings were made on a four-channel Grass Model 5C polygraph using a 5P5 preamplifier coupled to a driver amplifier for each channel. Recordings were made at a paper speed of 30 mm./sec. EEG activity was continuously monitored on a Tektronix Type 502 dual-beam oscilloscope.

Procedure.

Pretraining. Each S was trained to press a disc and to receive pellets from a delivery tray by an approximation method. Initially, a sheet-metal panel, approximately 18 in. square, was placed on the home cage. Centered in this training panel was an illuminable manipulandum identical to those on the display panel in the experimental apparatus. Also included were a pellet dispenser and delivery tray. Before introduction into the experimental chamber, the Ss were required to press the disc on this panel spontaneously and to eat the banana pellets.

In the experimental chamber, each S was trained to respond with both discs illuminated and the house lights off. A special neckplate with enlarged handholes was used until S was reliably pressing the discs, then the standard

neckplate was introduced and used for the remainder of the experiment. Magazine training was accomplished by offering pellets by hand, forcing S to reach through the handholes. Pellets were then placed in the delivery tray and the delivery mechanism was operated whenever S reached for a pellet. Disc pressing was then shaped by an approximation procedure, with low-level (0.5-1.0 ma.) goad shock applied to the tail as necessary to keep S active. Discrete trials were introduced when S's activity centered about the discs or, in the case of "fast" monkeys, as soon as pressing had begun. In discrete trials, one discriminandum was illuminated on each trial and the house lights were illuminated only between trials. Immediate responding was encouraged by brief applications of goad shock if S had not responded within 1-20 sec. of the start of a trial. Development of preferences for spatial (Left or Right) or nonspatial (Bright or Dark) cues was counteracted by covering each handhole, one at a time and in turn, for 15-30 min. periods. The position of the illuminated disc was randomized with the restrictions that a given disc was lit for no more than four consecutive trials and that each was illuminated an equal number of trials. In the late stages of discrete trial pretraining, continuous goad was given if S had not pressed within 20 sec. of a trial onset, and intertrial presses were automatically punished by a high-level shock which lasted for

the duration of the press. This 2.5-3.5 ma. shock was given in order to discourage pressing between trials. The shock also encouraged brief on-trial responses, for, if an S held a disc long enough for the selector relay which was operated by the programmers to flip from the on-trial to the intertrial position, the S would receive intertrial shock. All on-trial presses were rewarded, and electrode leads were attached when S was pressing readily on trials and minimally (less than four presses per interval) between trials. When these performance criteria had been met, the insoluble problem was introduced for six Ss, but four other Ss (Anton, Bengt, Gunnar, and Ingmar) were given CDP (10 mg./kg., mixed 10 mg./cc, p. o.) on one day, three to four days before the insoluble problem.

Insoluble Problem. During the insoluble problem, both handholes were open, and the set random sequence of right and left disc illuminations was maintained for 80 trials per day. The randomizer was used to insure that 50% of the responses were rewarded and 50% were punished and that no relation existed between reward or punishment and any of the available cues (Left, Right, Bright, or Dark). Goad shock was applied continuously for failure to press within 20 sec. of a trial onset until the press was made. A very high-level shock (4.0-4.5 ma.) was applied to the tail for 1.0 sec. as punishment for "incorrect" responses. The same shock was applied automatically for the duration

of an intertrial press and for failure to make a sufficiently brief on-trial press in order to discourage intertrial responding and on-trial disc-holding. On-trial pressing with both hands simultaneously was discouraged by a special circuit which allowed an earned reward to be superseded by an earned punishment. The special circuit delayed the reward pulse for 15 msec., allowing sufficient time for the on-trial punishment circuit to open a relay which prevented the pulse from reaching the delivery mechanism control. Insoluble problem sessions were given daily until S adopted a stereotyped response to one cue and used it exclusively for at least six consecutive days.

Ss receiving CDP were drugged 60 min. before the start of a session to insure absorption of the drug. CDP was given via stomach tube in a dosage of 15 mg./kg. (mixed 15 mg./cc). Placebos were not given because Feldman and Green (1966) reported that placebos had no effect on any aspect of responding when given to fixated squirrel monkeys in the soluble problem.

Soluble Problem. No drugs were given in this phase. The soluble problem required each S to make a nonreversal shift from its stereotype. Thus, if S was stereotyped to Left, then Dark was correct; if S was stereotyped to Bright, then Left was correct. The number of trials per day, the 20-sec. goad contingency, the daily testing, the set random sequence of disc illuminations, reward, and

punishment all remained unchanged. Experimentation ceased when the day-to-day level of response latencies, and the within-day pattern of latencies over the four 20-trial blocks, had been stable for three days, regardless of whether the stereotype had been maintained.

Electroencephalographic Recording.

Records of electrical activity in the amygdala and hippocampus were taken on the last 1-3 days of pretraining and on all succeeding days. Since it was not considered necessary, feasible, or economical to record for the entirety of each session, a sampling procedure was used. EEG records were taken continuously until the first 4-10 trials of a session had occurred and also for 4-10 of the last 20 trials of a session. The display on the dual-beam oscilloscope was continuously monitored in order to detect malfunctions in the recording system at times when EEG records were not being taken.

Histology.

Ss were anesthetized with Nembutal and sacrificed by perfusion through the heart with isotonic saline followed by 27% formalin. Sections 5-6 mm. wide containing the tracks of amygdala and hippocampal electrodes were removed and imbedded in paraffin. The tissues were then sliced into 25-u. sections, stained by the Kluver-Barrera technique, and examined for confirmation of the placement of electrode tips.

Results

Behavioral Results.

The experiment was conducted in two replications, with the six Ss in Group I beginning the insoluble problem four months prior to the four Ss in Group II (Table 1). In both Group I and Group II the Ss were paired for assignment (by coin-toss) to the drug or no-drug condition in the insoluble problem. Group I Ss were matched on the basis of sex and response latency during the last two days of pretraining. Group II Ss were matched on a more complex basis. One Group II S (Gunnar) could not be trained to use his right hand for disc pressing or even for taking reward pellets from the delivery tray, despite extended pretraining. Hence this S was considered stereotyped, was given only the soluble problem, and was assigned to the no-drug group. Of the three remaining Group II Ss, two were matched on the basis of a synchronous EEG rhythm to which attention had been drawn by the records of Group I Ss. Anton and Ingmar displayed more of this rhythm than Bengt, hence Anton and Ingmar were paired, and Bengt was matched with Gunnar by default and was assigned to the drug condition. Table 1 presents a summary of the groups.

Insoluble Problem. The insoluble problem was completed in 8-36 days by eight of the nine Ss to which it was given. Four drug-group Ss met the stereotype criterion of six consecutive days in which all on-trial presses were to a

Table 1. Assignment of monkeys to drug or no-drug conditions in the insoluble problem, with latency characteristics given for the last two days of pretraining.

CONDITION						
NO DRUG				DRUG		
Monkey	Mean Latency	Median of Mean Latency	Group	Monkey	Mean Latency	Median of Mean Latency
Frieda	5.3	2.4	I	Phaedra	7.9	8.6
Dodie	14.8	16.8	I	Winifred	13.5	15.0
Ulrich	20.4	19.8	I	Gustav	3.3	2.9
Anton	1.9	1.9	II	Ingmar	21.8	21.8
Gunnar	21.7	21.7	II	Bengt	22.6	22.6

single cue in an average of 18.5 days (median, 19.0 days), and the four no-drug Ss met the criterion in an average of 20.2 days (median, 18.5 days). Due to E's error, one no-drug S (Frieda) was given one extra stereotype day and one drug-group S (Phaedra) was given two extra stereotype days. For both Ss, the last six days were counted as criterional. The randomization test for two independent samples (Siegel, 1956, pp. 152-156) showed no difference between drug and no-drug groups with respect to days to criterion. (The nonparametric randomization test was considered more suitable than the parametric t-test for evaluation of data from this experiment because the randomization test is not as sensitive to atypical scores and does not require the assumptions of normally distributed scores and homogeniety of variance. In addition, power and efficiency of the randomization test are equivalent to those of the t-test.)

Since Bengt, the drug-group S not included above, had not adopted a stereotype by Day 38, it was decided to stop CDP administration on Day 39 and to continue the insoluble problem either for six days if no change in behavior resulted or for nine days if responding to the nonpreferred position decreased. A one-day reversal of the on-going Left preference occurred on Day 44, but was considered inconsequential because (1) the Left preference returned on Day 45 and (2) home-cage scratching had produced lesions

on the forearms in a place which contacted the handhole rims when presses were made, and since the lesion on the left was more severe than that on the right it was thought that the lesions could have determined the change in preference. Thus Bengt was given the soluble problem on the eighth day after CDP administration ceased.

Mean response latencies are compared in Fig. 1(A) for the four no-drug Ss and the four drug-group Ss who completed the six criterional days of the insoluble problem. One S (Dodie) in the no-drug group was consistently slow and accounted for that group's moderate mean latencies; one S in the drug group (Ingmar) was a moderate responder until the last two criterional days and accounted for the lower drug-group latencies on those two days. The randomization test for two independent samples showed no difference in mean latencies between drug and no-drug groups when the comparison was based on all six criterional days or when based on only the last two criterional days.

To overcome the effects of atypically slow Ss on the group means, group medians were examined (Fig. 1B). The median latency over the six criterional days was 1.4 sec. for the no-drug group and 2.1 sec. for the drug group. The difference was tested by comparing the group medians over the six criterional days using the randomization test for matched pairs (Siegel, 1956, pp. 88-92), and the difference barely missed statistical significance (p=.062,

Fig. 1. Mean latencies (A) and median latencies (B) of the drugged and non-drugged Ss during the six criterional days of the insoluble problem. Each point represents the data for a 20-trial block.

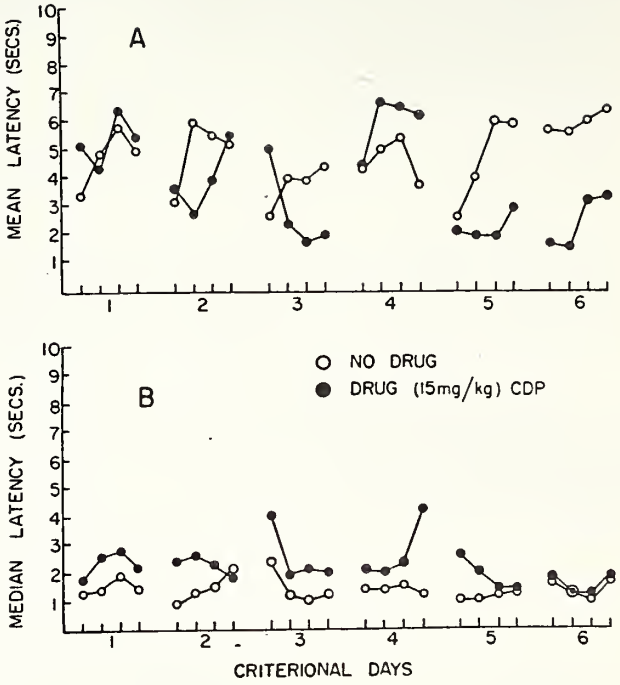

A
B
MEAN LATENCY (SECS.)
MEDIAN LATENCY (SECS.)
NO DRUG
DRUG (15mg/kg) CDP
CRITERIONAL DAYS

Fig. 1

two-tailed test).

A second means of evaluating the effects of CDP on response latencies was to compare mean latencies during the last two days of pretraining (Table 1) to mean latencies during the six criterional days of the insoluble problem (Table 2). The no-drug group displayed a mean decrease of 5.6 sec. (from 10.6 sec. in pretraining to 5.0 sec. in the insoluble problem) while the drug group displayed a mean decrease of 7.8 sec. (from 11.6 to 3.8 sec.). The presence of an atypical S in each group (Ulrich in the no-drug and Gustav in the drug group -- see Table 2) prevented the difference in mean decreases from being significant when tested with a one-tailed randomization test for independent samples. The changes in mean latencies from pretraining to the insoluble problem were also tested for each group separately using one-tailed randomization tests for matched pairs. The mean decrease of the no-drug group was not significant ($p>.10$), but the mean decrease of the drug group was as significant ($p=.062$) as the small number of Ss ($n=4$) would allow.

Records of intertrial presses were examined for both groups and a comparison was made over the six criterional days. The mean number of intertrial presses per day for the no-drug group ($n=4$) was 18.4 while that for the drug group ($n=4$) was 251.2. Despite extreme intersubject variability (Table 2), the randomization test for independent

Table 2. Response data for the four Ss in the no-drug and drug groups which met the stereotype criterion in the insoluble problem over the six criterional days. Criterional days, mean latencies, change in mean latency from pretraining, and mean intertrial presses per day are shown.

Group	Monkey	Criterional Days	Mean Latency	Latency Change from pretraining	Mean Intertrial Presses
No Drug	Frieda	3-8	1.8	-3.5	36
	Dodie	14-19	16.6	+1.8	13
	Ulrich	13-18	0.8	-19.6	7
	Anton	31-36	0.8	-1.1	38
Drug	Phaedra	7-12	2.3	-5.6	27
	Winifred	21-26	0.5	-13.0	1099
	Gustav	6-11	2.3	-1.0	102
	Ingmar	22-27	10.2	-11.6	20

samples yielded a probability of p=.058 for this difference. Group medians were examined to remove the inflationary effects of individual Ss on the results. Over the six criterional days the median intertrial presses per session were 20.2 for the no-drug group and 46.2 for the drug group. A one-tailed randomization test for matched pairs was used to evaluate the difference between group medians over the six criterional days and yielded significance at the p=.031 level.

Upon examination of the number of days in which a stereotype was practiced prior to the six criterional days, it was found that the number of such days was 18 for the no-drug Ss and 22 for the drug Ss (Table 1, Appendix). In each group, two Ss had one or no stereotype days prior to the criterional days and two Ss had seven or more. The randomization test for independent samples showed the difference to be not significant.

Soluble Problem. The soluble problem was given to all Ss with Dark correct because all Ss had adopted position stereotypes.

In the soluble problem only one S, Phaedra, a drug-group S, deviated more than seven times from its stereotyped response. She first responded to the non-stereotyped position on Day 3, and until meeting the termination criterion (of three consecutive days with mean latencies at the same level and distributed within days in the same pattern) on

Day 14, she deviated on a total of 11 days. On eight days no more than four deviant presses were made, but on Days 12, 13, and 14 deviant presses were made 35, 48, and 36 times, respectively. Deviant presses were not necessarily made when Dark, the correct stimulus, was in the position opposite the stereotype. For example, on Day 14 the percentage of correct responses was 61 while a maximum of 95% correct was possible with 36 deviant presses. Phaedra was run for an additional 25 days to encourage solution of the problem, but no day was obtained in which performance exceeded 80% correct.

Bengt, the drug-group S for whom the stereotype criterion was waived in the insoluble problem, adopted a stereotype to Right on the first two days of the soluble problem and maintained the stereotype throughout the remainder of the experiment (Table 1, Appendix).

The termination criterion was met in 3-36 days. Both extremes occurred in the no-drug group (Table 3), but the means were nearly identical at 14.8 and 14.6 days for the no-drug and drug groups, respectively, and a randomization test for independent samples showed that the groups did not differ in this respect.

All comparisons of latencies in the soluble problem were based either on the three days required for meeting the termination criterion (three criterional days) or on those days plus the three days which immediately preceded them

Table 3. Response data for all Ss during the three criterional days of the soluble problem. Criterional days, mean response latencies to correct and incorrect ones, and mean intertrial presses per day are shown.

Group	Monkey	Criterional Days	Mean Latency Correct	Mean Latency Incorrect	Mean Intertrial Presses
No Drug	Frieda	6-8	0.9	1.0	9
	Dodie	13-16	18.9	17.3	4
	Ulrich	1-3	0.8	0.7	6
	Anton	9-11	0.7	0.7	85
	Gunnar	34-36	17.6	16.3	80
Drug	Phaedra	12-14	0.9*	0.9*	64
	Winifred	4-6	0.6	0.6	325
	Gustav	8-10	0.7	0.9	317
	Ingmar	19-21	1.5	1.4	7
	Bengt	16-18	1.2	1.2	212

*Because the semi-automated apparatus was not equipped to record latencies of responses more specific than position responses, and because Phaedra did not practice a stereotyped position response on these days, the means represent overall mean latencies. However, there is no reason to believe that Phaedra's latencies at this time were differential (Table 1, Appendix).

(six terminal days). Comparisons based on the three criterional days include all Ss, but comparisons based on the six terminal days do not include Ulrich, the no-drug S who met the termination criterion on the first three days of the soluble problem.

Figs. 2(A) and 2(B) show the mean and median latencies of the drug and no-drug groups to correct and incorrect stimuli on the terminal six days. Ulrich's data are included in the means and medians of the no-drug group for illustrative purposes only: the drop in latencies in the no-drug group indicates that he was a fast responder, and the lack of change in the latency difference between correct and incorrect stimuli shows that Ulrich, as well as the other Ss in the group, failed to respond differentially to correct and incorreot. Two-tailed randomization tests for matched pairs were used to evaluate the differences in latencies between correct and incorrect stimuli in the no-drug group and in the drug group. Neither the tests over the six terminal days (with Ulrich excluded from the no-drug comparison) nor the tests over the three criterional days (with Ulrich included) yielded significance for either group. Since there was no latency difference between stimuli for either group, latencies were pooled over stimuli for a comparison of the latencies of the two groups. Randomization tests for independent samples were used to evaluate the means and randomization

Fig. 2. Mean (A) and median (B) response latencies of the no-drug and drug groups to correct and incorrect stimuli during the six terminal days of the soluble problem. Each data point represents mean or median latency to correct or incorrect during a 20-trial block. Ulrich's data are included in the no-drug group's results on only the last three days (for explanation, see text).

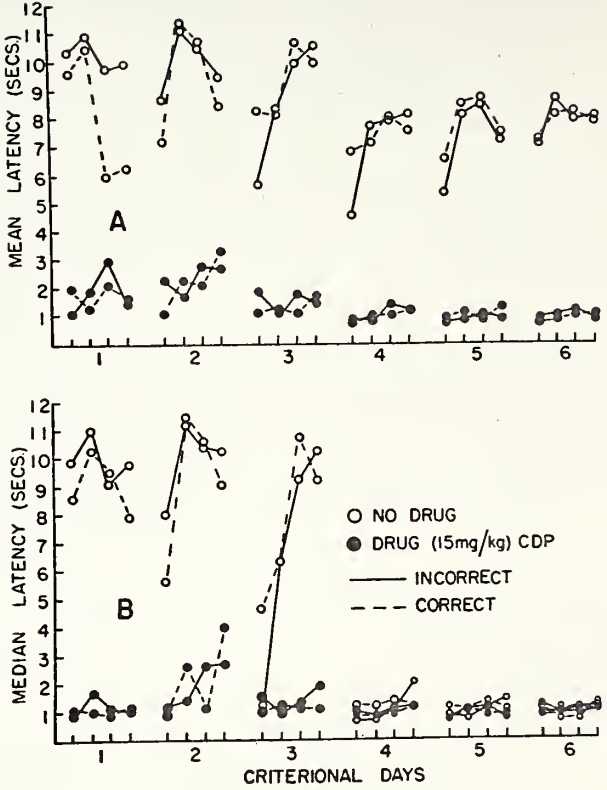

MEAN LATENCY (SECS.)
A
MEDIAN LATENCY (SECS.)
B
O NO DRUG
● DRUG (15mg/kg) CDP
INCORRECT
CORRECT
CRITERIONAL DAYS

Fig. 2

tests for matched pairs were used to evaluate the medians over the six terminal days and over the three criterional days. Not one of the four tests yielded significance.

Intertrial presses were compared for the two groups (Table 3). Over the six terminal days the mean daily number of intertrial presses was 44.6 for the no-drug group and 159.1 for the drug group. A one-tailed randomization test for independent samples showed this difference to approach significance ($p=.079$). Over the three criterional days the no-drug group averaged 36.7 intertrial presses per day and the drug group averaged 185.2. This difference was significant at the $p=.052$ level.

To check the possibility that this difference might have been due to persistence of CDP, intertrial presses were examined in the records of three drug-group and one no-drug Ss which were given the soluble problem for at least eight additional days after meeting the termination criterion. One-tailed randomization tests for matched pairs were used to compare intertrial responding on the terminal days to that on six post-termination days in a block beginning on the third day after criterion was met. Gustav and Phaedra both displayed significant decreases from 215.3 to 3.0 mean daily intertrial presses ($p=.016$) and from 61.1 to 25.1 ($p=.031$), respectively. However, Winifred's scores displayed an increase (from 343.9 to 583.2) which was not significant. Winifred's increase was due to

elevated responding on the first three days of the post-termination block; intertrial responding on the last three days of the block decreased to an average of 168.7 presses per day. The comparison for the sole no-drug S (Frieda) showed that the decrease from 13.5 to 9.5 was not significant.

Ancillary Observations. Informal observations revealed two additional effects of CDP. One drug-group S (Phaedra) vocalized and violently resisted being placed in the restraining chair during pretraining and during the soluble problem, but during the insoluble problem, when CDP was given, the vocalization and struggling did not occur. A second effect was acceptance of reward pellets. Two Ss (Phaedra and Ingmar) tended to accept pellets more often under CDP than in the non-drugged state, but another S (Gustav) accepted pellets less often under CDP. The remaining Ss in the drug group showed no remarkable contrast between drugged and non-drugged states, and no Ss in the no-drug group behaved differently in one phase than they did in any other phase.

Electroencephalographic Results.

A signal was observed which was characteristic of some sites in the amygdala and hippocampus (Fig. 3A). The signal was a regular 10-14/sec. (usually 11-13/sec.) rhythm which was 50-500 uV. in amplitude and was displayed in one or more leads from Frieda, Dodie, Anton, and Gunnar in the

Fig. 3. Sample recordings from Phaedra. (A) Rhythmic activity without drug. (B) Rhythmic activity one hour after a dosage of CDP (30 mg./kg.). Traces, from top to bottom, represent activity in the left amygdala, events and time in sec., activity from the right amygdala, activity from the left hippocampus, and activity from the right hippocampus; amplitude markings represent 50 uV. Records from other animals displayed rhythmic activity from one or more sites, and not necessarily from all four as in Phaedra's case.

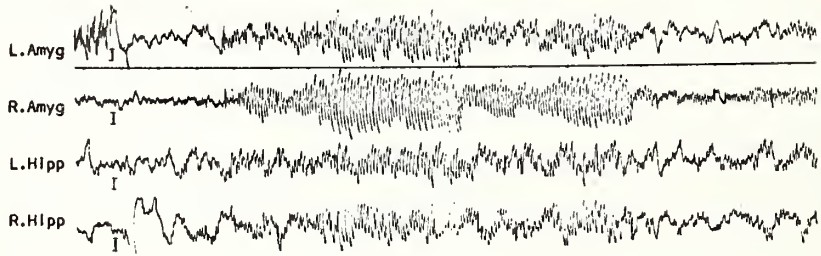


Fig. 3

no-drug group and from Phaedra, Ingmar, and Bengt in the drug group. The range in frequency was due mainly to differences between Ss: the rhythm was constant within 1-2/sec. in records from a given site, but considerable variation in amplitude could be observed from any given lead. Incidental observation indicated that the rhythm occurred only when the Ss were motionless, usually with eyes closed or half-open, teeth slightly bared, and arms held out in front of them contacting nothing.

Recordings were made continuously until 4-10 trials had elapsed during the first 20-trial block and again during the last 20-trial block of each session. If the rhythm occurred in recordings from a given lead, then all recorded activity from that lead was scored, except activity prior to Trial 1 of a session and activity which was obscured by flexion artifact or some other interference (such as 60/sec. noise, recovery of the polygraph from shorting of the leads during a punishment, etc.). In short, then, all scorable activity from a channel in which the rhythm occurred was scored.

Criteria for identification of a burst of the rhythm were as follows: (1) the rhythm had to fall within the frequency range of 10-14/sec.; (2) the rhythm had to be regular, that is, the peaks or troughs had to be temporally equidistant for the duration of the burst; (3) the rhythm had to occur for at least 0.6 sec.; and (4) the

amplitude of the waves had to be at least 20 uV. greater than the amplitude of the immediately preceding and succeeding activity. It was considered desirable that the rhythm appear to be sinusoidal in character, but this extreme occurred so seldom that sinusoidal character was not a scoring criterion.

When scoring the records of each S for each session, the total amount of scorable recorded time in each intertrial and each on-trial interval was noted to the nearest second, the total amount of time that the rhythm occurred in each interval was noted to the nearest second, and the number of bursts within each interval were noted. Since differences in the amount of scorable recorded time occurred from S to S as well as from day to day within Ss, three ratios were devised to characterize the rhythm's occurrence. These ratios were the percentage of time that the rhythm occurred within each type of interval (intertrial or on-trial), the mean duration of bursts within each interval, and the mean amount of time between initiations of bursts within each type of interval. The last ratio was partially dependent upon mean duration and yielded highly variable data, hence it was not used for analysis. Ratios were calculated for intertrial and on-trial intervals separately.

Attempts to relate rhythmic activity to reward or punishment failed, which may be consistent with the lack

of latency difference between correct and incorrect stimuli in the soluble problem. As can be seen in Table 2 of the Appendix, no consistent difference in the amount of rhythmic activity occurred in any lead of any S after reward or after punishment. Hence the post-punishment and post-reward data were pooled, and all comparisons which follow were based on the pooled data.

The occurrence of the 10-14/sec. rhythm in the insoluble problem was compared to that in the soluble problem in order to evaluate the effect of CDP on the rhythm. All comparisons were based on the data of individual Ss because individual differences were too extreme to allow between-groups comparisons and because the small number of Ss prevented acceptable significance levels from being attained (i. e., with n=3, the best significance level obtainable with the randomization test for matched pairs is p=.125) in within-groups comparisons. Comparisons were based on the intertrial data from the six terminal days of each problem for each S. Only two Ss had on-trial latencies great enough to allow on-trial EEGs to be obtained, and since one of these (Gunnar) was not given the insoluble problem, on-trial comparisons were not made. Since a preamplifier failure prevented data from being collected from some Ss for some of the six terminal days, only pairs of days for which data were available were used for analysis. One-tailed randomization tests for matched pairs were used

for comparisons on all drug-group Ss because there was reason to believe that CDP reduced the rhythmic activity. Two-tailed tests were used for all no-drug comparisons.

The 10-14/sec. rhythm was found in eight amygdala placements, the records from which were free from interference sufficiently often to allow scoring, and the summary of the results from these leads is shown in Table 4. In terms of the percentage time measure, one no-drug S (Frieda) displayed a significant decrease ($p=.031$) from insoluble to soluble problem but two other no-drug Ss did not change. In the drug group, neither lead from Phaedra changed, but Ingmar and Bengt both displayed significant increases ($p=.016$ and $p=.031$, respectively). Bengt's EEG data were taken from the six days immediately preceding removal of CDP because the stereotype criterion was waived for this S. This procedure maintained the confounding of terminal drug days and criterional stereotype days which was characteristic of the other drug-group Ss, hence the comparisons between insoluble problem and soluble problem may be viewed as evaluations of the effect of CDP. To summarize results based on the percentage time measure, then, one no-drug S displayed a significant decrease and two drug-group Ss displayed significant increases from insoluble to soluble problems; the remaining Ss showed no change.

Comparisons were also made for individual Ss using mean

Table 4. Characteristics of the occurrence of the 10-14/sec. rhythm during the six terminal days of the insoluble and soluble problems. Abbreviations:

GP=Group Amyg=Amygdala Insol=Insoluble Problem
ND=No Drug Hipp=Hippocampus Sol =Soluble Problem
D=Drug
Prob=Probability No=Number of Pairs of Days

			Percentage Time			Burst Duration			
GP	Monkey	Site	Insol	Sol	Prob	Insol	Sol	Prob	No
ND	Frieda	LAmyg	21.6	14.7	.03	1.4	1.2	>.05	5
		LHipp	9.8	5.4	.03	0.8	0.9	>.05	6
		RHipp	8.8	6.3	>.05	1.0	1.0	>.05	6
ND	Dodie	RAmyg	12.0	13.9	>.05	1.1	1.2	>.05	3
		LHipp	9.5	20.9	.03	0.8	0.9	>.05	5
ND	Anton	RAmyg	2.7	2.5	>.05	1.0	0.9	>.05	6
ND	Gunnar	RAmyg	--	0.7		--	1.0		6
D	Phaedra	LAmyg	27.4	34.9	>.05	1.7	2.8	>.05	4
		RAmyg	37.2	34.2	>.05	2.3	2.6	>.05	2
		LHipp	14.3	15.8	>.05	1.3	1.2	>.05	6
		RHipp	20.2	25.4	>.05	1.5	1.5	>.05	6
D	Ingmar	RAmyg	13.5	27.1	.02	1.1	1.7	.02	6
D	Bengt	RAmyg	0.5	8.8	.03	0.9	1.1	>.05	6

burst duration as the basic datum (Table 4). In the no-drug group, no S displayed a significant change, while in the drug group Ingmar displayed a significant increase ($p=.052$). Changes which were not significant in the no-drug group were also not consistent in direction, while in the drug group all amygdala leads displayed increases. Thus, in terms of mean burst duration the no-drug group again showed no consistent change, while a consistent increase occurred in the drug group, but with an acceptable significane level in only one S.

The foregoing comparisons suggest that CDP has a greater influence than problem over the occurrence of the 10-14/sec. rhythm, at least in terms of records from the amygdala. To explore the issue further, one-day experiments were conducted where recordings were made before and after CDP administration. Anton, Bengt, Gunnar, and Ingmar were given this treatment in pretraining, and Phaedra received it after completion of the experiment. The randomization test for matched pairs, when appled to the percentage time measure, showed the decrease from 19.7% to 7.5% to be significant at the $p=.031$ level. However, the decrease in mean burst duration from 2.2 to 1.2 sec. was not significant ($p=.062$) because Gunnar's duration did not change. A further test of the issue was made possible by the fact that Bengt was given seven insoluble problem sessions after CDP administration ceased. The last seven days with CDP (Days 32-38) were compared to the seven insoluble prob-

lem days on which no CDP was given (Days 39-45). Days were matched symmetrically-about the drug-removal point, e. g., Days 38 and 39 were paired, Days 37 and 40 were paired, and so on. The increase in percentage time from 0.5 to 7.6 sec. was significant at the p=.016 level. However, the increase in mean burst duration from 0.9 to 1.0 sec. was not significant due to identical durations on three pairs of days.

The 10-14/sec. rhythm was found in five hippocampal sites (Table 4). Comparisons were made on these records in the same fashion as the comparisons above, except that two-tailed tests were used for the drug-group S (Phaedra) as well as for the two no-drug Ss (Frieda and Dodie). In terms of the percentage time measure, both no-drug Ss showed significant changes, but in opposite directions: Frieda decreased in both leads, but significantly only in the left hippocampus (p=.031); Dodie displayed an increase which was significant at the p=.031 level. Both leads from the drug-group S increased, but neither was significant. In terms of mean burst duration, both Frieda and Dodie displayed an increase, but neither was significant; the drug-group S displayed a nonsignificant decrease in one lead and no change in the other lead.

Rank-order correlations were used to relate rhythmic activity in the hippocampus to that in the amygdala during the insoluble problem and during the soluble problem in

Table 5. Rank-order correlations between amygdala and hippocampus based on percentage time of occurrence of the 10-14/sec. rhythm.

I N S O L U B L E P R O B L E M

Group	Monkey	Sites	rho	No. Days	Prob.
No Drug	Frieda	LAmyg-LHipp	-.13	8	
		LHipp-RHipp	+.67	8	<.05
	Dodie	RAmyg-LHipp	+.24	15	
Drug	Phaedra	LAmyg-LHipp	+.76	11	<.01
		RAmyg-RHipp	+.50	11	
		LAmyg-RAmyg	+.78	11	<.01
		LHipp-RHipp	+.75	11	<.01

S O L U B L E P R O B L E M

Group	Monkey	Sites	rho	No. Days	Prob.
No Drug	Frieda	LAmyg-LHipp	+.80	6	
		LHipp-RHipp	+.51	7	
	Dodie	RAmyg-LHipp	-.76	9	
Drug	Phaedra	LAmyg-LHipp	+.67	12	<.05
		RAmyg-RHipp	+.52	8	
		LAmyg-RAmyg	+1.00	6	<.01
		LHipp-RHipp	+.90	14	<.01

the three Ss from which scorable activity was observed in both structures. Only the percentage measure was used because the duration measure had too many ties to allow valid correlations to be made. As can be seen in Table 5, the only correlations which achieved significance were those between Phaedra's left amygdala and hippocampus. The correlations between these ipsilateral structures were positive both in the insoluble problem (rho=0.76, $p<.01$) and in the soluble problem (rho=0.67, $p<.05$). Correlations between Phaedra's left and right amygdala and left and right hippocampus, as well as between Frieda's left and right hippocampus are included in Table 5 to allow evaluation of the relationship between pairs of structures during both phases of the experiment.

Histological Results.

Confirmation was made of the sites of the tips of 15 electrodes directed towards the amygdala and of 17 electrodes directed towards the hippocampus. Of the five unconfirmed amygdala placements, two are accountable to Gustav, who died after the experiment and was destroyed; two are accountable to Ulrich, whose cement cap loosened with the electrodes, was unwittingly replaced, and was found to have damaged extensive portions of the tissue around the amygdala; and one was due to poor histology on Dodie's left amygdala. Rhythmic activity was not observed from any of these sites. Of the three uncon-

firmed hippocampal placements, two are accountable to Gustav, and one is accountable to poor histology on Dodie's left hippocampus. Unfortunately, rhythmic activity was observed from Dodie's left hippocampus.

Occurrence of rhythmic activity is related schematically to sites of electrode tips in the amygdala in Fig. 4(A) and in the hippocampus in Fig. 4(B). The anterior-posterior (AP) planes in which the tips were located were determined by reference to the atlas of Gergen and MacLean (1962). The average amygdala placement was 9.4 mm. anterior from the interaural line, and the actual placements deviated less tha 1.0 mm. anterior and slightly more than 1.0 mm. posterior from the mean. The representation of amygdala sites in Fig. 4(A) is a composite with gross structure based on plane AP 9.5 of the Gergen and MacLean atlas and with amygdala nuclear groups based on the equivalent section in the Emmers and Akert (1963) atlas. Thirteen placements are represented. Winifred's two confirmed sites are omitted because hyperactivity and excessive intertrial pressing prevented evaluation of her EEG records. One site from which unscorable rhythmic activity was noted is shown: the records from Frieda's right amygdala were obscured by interference on too many days to allow meaningful scoring. The sites shown suggest that rhythmic activity derived from the posteromedial portions of the amygdala, includ-

Fig. 4(A). Representation of sites of electrode tips in the amygdala. Circles represent sites of rhythmic activity. Crosses represent no rhythmic activity. Abbreviations:

Aq	Aqueduct	IC	Internal Capsule
BA	Basal Accessory Amygdala	INF	Infundibulum
BLA	Basal Lateral Amygdala	It	Nucleus Intercalatus
BMA	Medial Basal Amygdala	LA	Lateral Amygdala
CeA	Central Amygdala	MA	Medial Amygdala
GP	Globus Pallidus	II	Optic Tract

Fig. 4(B). Representation of sites of electrode tips in the hippocampus, with circles representing sites of rhythmic activity and crosses representing sites of no rhythmic activity. Abbreviations:

Aq	Aqueduct	Hipp	Hippocampus
CC	Corpus Callossum	Put	Putamen
Ci	Cingulate Gyrus	Re	Reticular Substance
CSp	Corticospinal Tract	IV N	Nucleus of Trochlear Nerve
ColS	Superior Colliculus	III	Third Ventricle

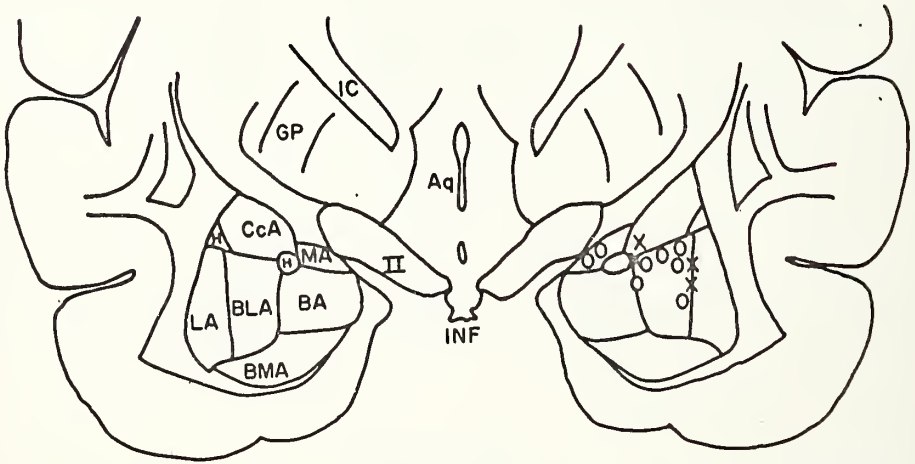
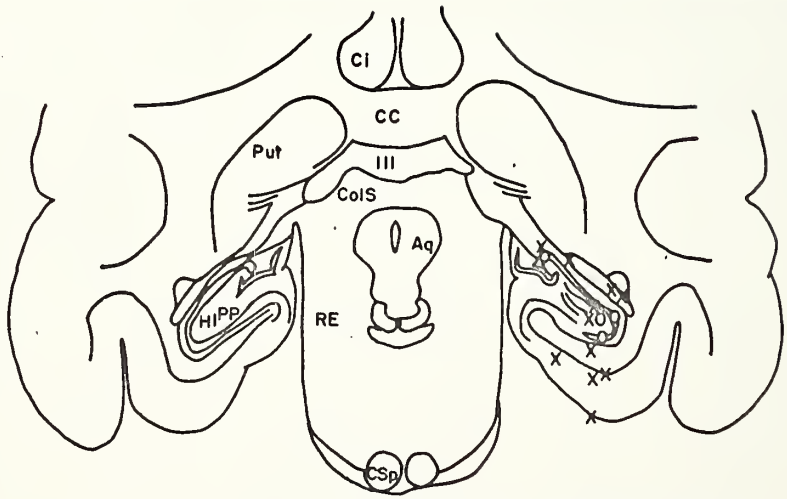

IC
GP
Aq
CcA
MA
II
LA
BLA
BA
BMA
INF
O = SITE OF 10-14/SEC. RHYTHM
X = NO 10-14/SEC. RHYTHM

Fig. 4 (A)



O = SITE OF 10-14/SEC. RHYTHM

X = NO 10-14/SEC. RHYTHM

Fig. 4 (B)

ing basal lateral, basal accessory, intercalated, and medial nuclear groups.

Fig. 4(B) schematically depicts sites in terms of occurrence and non-occurrence of rhythmic activity in the hippocampus. Variations about the AP plane depicted (AP 1.5 from the atlas of Gergen and MacLean) were less than 1.0 mm., and the representation is based entirely upon the atlas of Gergen and MacLean. Winifred's two confirmed sites are not included, for reasons described above. Rhythmic activity from the posterior hippocampus appeared to derive from the pyramidal and stratum radiatum layers.

Discussion

The main findings of the experiment were as follows:
(1) all Ss but one adopted a stereotyped response in the insoluble problem:
(2) all Ss but one maintained their stereotyped responses throughout the soluble problem, and the one S which deviated did not solve;
(3) the Ss which received CDP displayed a tendency towards elevated intertrial responding in the insoluble problem, and the elevation persisted through the terminal days of the soluble problem;
(4) the Ss receiving CDP displayed decreased latencies in the insoluble problem; and
(5) the occurrence of a regular 10-14/sec. rhythm, which was typical of some sites in amygdala and hippocampus, was found in amygdala records to decrease in Ss given CDP.

That 9 of 10 monkeys adopted stereotyped responses in the insoluble problem was to be expected on the basis of previous results (Feldman & Green, 1966), but the total failure of Bengt to stereotype within 45 days is surprising. Conceivably, CDP could exercise a stereotype-preventive effect, but Bengt's behavior during the eight days after CDP was removed and the lack of reports in the rat literature of similar failures to stereotype (Feldman, 1962; 1964a; 1964b) do not support such a notion. It appears that the noncriterional stereotype which Bengt

adopted and practiced in the soluble problem was due mainly to the lesions on his forearms, although there is no way to rule out the possibility that CDP had some influence.

At this point attention should be called to a methodological artifact which could have influenced many aspects of responding in this experiment and which was not present in the experiment of Feldman and Green (1966). The artifact was a confounding of clicks of the relays which were mounted on the test chamber with onset and offset of punishment shock. With regard to intertrial presses, a click and shock onset were correlated with the press of a disc, and click and shock offset were correlated with release of the disc. On a punished trial, a click and onset of the 1.0-sec. punishment shock were correlated with the disc press, but, since Ss usually released the disc as a reflex to the onset of shock, the click associated with shock offset was independent of disc release. Occasionally on punished trials, an S would hold the disc slightly longer than was necessary for the programmer to be activated. As a result, the selector relay which was operated by the programmer would flip to the intertrial position and would activate the circuit for intertrial shock. However, since intertrial and on-trial punishment shock were taken from the same generator, and since S generally released the disc prior to offset of the 1.0-sec.

punishment shock, the only result of possible disc-holding on a punished trial was the addition of one press to the total on the intertrial press counter.

Possible implications of the confounding of clicks with shock and disc presses in intertrial and punished-trial responding are (1) that relay clicks could have become cues indicating that S had pressed with sufficient vigor to activate an intertrial or on-trial event or (2) that relay clicks could have acquired reinforcement value because the second of every pair of clicks was always associated with termination of shock. Hence in the case of a rewarded trial, where a properly-executed press produced no sound other than the buzz of the delivery mechanism, the Ss of the present experiment might have been encouraged to hold the discs in order to obtain the clicks whereas the Ss in the experiment of Feldman and Green (1966), although they were noted to hold the discs occasionally, had no such encouragement. An S which held a disc on a rewarded trial not only was presented with a pair of clicks, but also received a shock. Disc-holding on a rewarded trial, then, yielded one click for shock onset after the actual press had been made, and a second click for shock offset which was correlated with release of the disc. The shock received in this manner would be brief because Ss reflexly released the disc, but the presentation of the clicks would be effected (along with whatever information or reinforcement

value which might have been associated with them), and one press would be added to the total on the intertrial press counter. It can clearly be seen that an S which was encouraged to hold the response disc would receive shock in addition to food on rewarded trials, and that the presentation of clicks and punishment on both rewarded and punished trials would make differentiation between correct and incorrect stimuli very difficult in both the insoluble and soluble problems. It can also be seen that disc-holding would inflate intertrial pressing scores.

Evaluation of the frequency of disc-holding across or within Ss is difficult because the temporal distribution of intertrial presses was not recorded. It was E's impression that many intertrial shocks were obtained in the manner just described. However, since the actual effects of the relay-click artifact vis-à-vis disc-holding cannot be determined, the possible effects must be considered when interpreting the results which follow.

If squirrel monkeys were affected by CDP in the present situation in the same way as rats on a Lashley jumping stand, three or four of the five drug-group Ss would have been expected to solve, since Feldman (1962) reported that 73% of rats given CDP in the insoluble problem solved the soluble problem. Only one of the five drugged monkeys even broke its stereotype, but since this S was in the drug group the result is at least not contrary to expecta-

tion. The artifact just discussed could have played a very powerful role in the failure to obtain solutions, for, as described above, it could have complicated the task of differentiating between correct and incorrect stimuli. Indeed, the only S which broke its stereotype (Phaedra) never solved, and it can be shown that her better performance in terms of percentage correct responses might have been related to reduced frequency of intertrial presses. The 39 soluble-problem days (14 until meeting criterion and 25 additional days) of this S were examined and assigned to the four categories which follow (out-off points were selected so as to make the number of days in each category as equal as possible): with regard to intertrial presses, (1) days with more than 50 (n=11) and (2) days with less than 12 (n=13); with regard to percentage correct responses, (3) days where performance was 60% correct or better (n=11) and (4) days where it was 52.5% or worse (n=12). The following 2 X 2 contingency table was then constructed with cell entries being days which met the four criteria obtained by crossing the intertrial press extremes with the performance extremes.

		Intertrial Presses	
		>50	<12
Percent Correct Responses	>60.0%	2	7
	<52.5%	6	1

The negative relation between percentage correct responses

and intertrial presses was found by the Fisher exact probability test (Siegel, 1956, pp. 96-104) to be significant ($p<.025$, one-tailed test).

This demonstration would be a more convincing indictment of the effect of the artifact if it were known that all intertrial presses were due to disc-holding. But because the temporal course of intertrial presses was not recorded, the role of disc-holding on this aspect of soluble-problem performance is not as clear as could be desired. It is possible that a persisting effect of CDP could have interfered with the soluble-problem performance of this S, for squirrel monkeys will show an effect on performance in a non-discriminated avoidance task one week after a single dose (10 mg./kg.) of CDP.[2] However, since Feldman (1962) reported that rats given CDP during both phases of the Maier paradigm were able to solve, it seems unlikely that a persisting effect of CDP could alone have prevented Phaedra from solving. More likely, the failure was due to a combination of persisting effects of CDP and of the effects of the relay-click artifact.

Although the drug group did not display mean or median latencies lower than the no-drug group, they did display a greater decrease in latencies from the end of pretraining to the criterional days of the insoluble problem. All

2. Dr. C. L. Scheckel, personal communication, 1966.

four drug-group Ss displayed decreases, and the decreases of three of the four were large. On the other hand, only three of the drug Ss displayed decreases, and the decrease of only one S was large. Hence the present results appear to be in line with results obtained with rats (Feldman, 1962). The failure to obtain significance in the test of the drug group's decrease was due to the small number of Ss, and if the last six drug days of Bengt are included in the comparison between latencies in pretraining and in the insoluble problem, the resulting decrease is significant at the p=.032 level.

It is unclear what effect, if any, the relay-click artifact or the subsequent possibility of disc-holding could have had on group latencies in the insoluble problem. It might be argued that if the relay-click artifact had induced all Ss to hold the response discs then, since punishment shock would be given on every trial, all Ss would ignore the stimuli and would wait until onset of goad shock before pressing. The experiment might then resemble a nondiscriminated avoidance situation, with intervals between goad shocks representing shock-shock intervals. This set of circumstances, occurring early in the insoluble problem before an S's responding had stabilized, might induce a general increase in response frequency which would result in increased intertrial response totals and in decreased but highly variable on-trial latencies. Sidman

(1960) described an analogous situation and pointed out that "elimination of the warning stimulus was found to result in an increased rate of avoidance responding. The animals no longer waited until a shock was imminent before pressing the lever, but responded much more rapidly than was necessary" (pp. 302-303). However, if a mechanism such as this was operating, then the latencies of the drug-group Ss should have remained high or increased, and their intertrial press scores should have remained low. These expectations are based upon the observations of Hanson (1961) and of Vernier, Hanson, and Stone (1962) that squirrel monkeys given a tranquilizer (chlorpromazine) in a nondiscriminated avoidance situation either failed to change their rate of avoidance responding or actually decreased. Thus a nondiscriminated avoidance interpretation appears to be inadequate as an explanation of the decreased latencies of the drug group during the insoluble problem in the present experiment. Nor is such an interpretation consistent with the failure of Dodie and Gunnar in the no-drug group to display a decrease in latencies (Table 1, Appendix).

The failure to obtain differential latencies to correct and incorrect stimuli in the soluble problem was unexpected on the basis of experiments with rats (Feldman, 1964a; 1964b). It is possible that differences in species and situations may account for the failure to obtain dif-

ferential latencies. At first glance it appears that support is offered this interpretation by the results of Feldman and Green (1966) who found that squirrel monkeys which displayed significantly differential latencies with CDP in the soluble problem did not differentiate as greatly on no-drug days. However, Feldman and Green attributed their failure to obtain differential latencies without the drug to an atypical S which responded much faster to the incorrect than to the correct stimulus and thereby negated the more typical differential latencies of the remaining Ss. In the present experiment no S in either group consistently displayed differential latencies (Table 1, Appendix). The possibility that the relay-click artifact could have produced disc-holding which disrupted differentiation between correct and incorrect stimuli thus appears quite plausible. The complication of the task of differentiation which disc-holding could have produced could very well underlie the failure to obtain differential latencies, for, as described above, an S which held the disc would receive shock on rewarded as well as on punished trials. Possibly related to this effect was another factor which was noted in casual observation of the Ss. This factor was the failure of any S to observe the two discs systematically prior to each response. Only two Ss, both in the drug group, were observed to look at both discs. Phaedra looked at the disc on her stereotyped

side infrequently, and when she did look, it was after initiation of the press. If the incorrect cue was in the stereotyped position at that time, then she would look to the other side at the correct cue but would not stop the press. The second S, Bengt, occasionally looked from one disc to the other, principally during the insoluble problem, but was seldom reinforced because he generally pressed with both hands simultaneously and therefore received punishment due to the special circuit included to discourage simultaneous pressing. A disc-holding S, by virtue of receiving punishment on a large percentage of trials, would be discouraged from learning to observe the discs before pressing, because, as Stollnitz (1965) points out, "an observing response is still a link in a chain leading to the response on which reinforcement is based (the effective response). So acquisition and extinction of observing responses are determined by the same variables that determine acquisition and extinction of other chained responses" (p. 249).

With regard to intertrial presses per se, during the criterional days of the insoluble problem the drug-group Ss displayed a tendency towards more intertrial presses than the no-drug Ss. That CDP alleviates suppression of punished responses has been found by Feldman and Green (1966) in a comparison of intertrial presses of squirrel monkeys on drug and no-drug days and by Geller who found

that cynomolgous monkeys (1962) and rats (1964) would accept more shock with CDP when barpressing for food than without CDP. The possible disc-holding effect of the relay-click artifact could have contaminated the present results by inflating the intertrial press scores, as was described earlier. It is possible that disc-holding would be more likely to occur in drugged than in non-drugged Ss because the 15 mg./kg. dosage of CDP made the Ss slightly ataxic. However, reference to Table 2, which shows the mean daily intertrial press scores over the six criterional days of the insoluble problem for each S, does not clearly support this possibility because two no-drug Ss (Frieda and Anton) had intertrial press scores which were greater than those of two drugged Ss (Phaedra and Ingmar). Possibly, the relay-click artifact could have accounted for all intertrial presses in both groups up to a maximum of 80 (the number of trials in a session) by encouraging on-trial disc-holding. If this possibility is considered and 80 is subtracted from each score in Table 2 (with scores 80 or below subtracted to zero), then only two Ss (Gustav and Winifred) remain with any intertrial presses at all. Although the two Ss are in the drug group, which is consistent with past results, the difference between groups is no longer close to significance. In lieu of definitive means of evaluating the time or frequency of occurrence of disc-holding, the more conservative con-

clusion that disc-holding accounted for the observed intertrial press results might be warranted. However, in light of the fact that the present results are in line with past findings, the less conservative assumption that both groups were affected equally by the artifact may be made, and the interpretation favored is that the observed difference reflects a genuine effect of CDP.

With regard to intertrial presses in the soluble problem, a difficulty is presented by the fact that the difference between drug and no-drug groups persisted through the terminal days, long after CDP administration had stopped. The possibility that disc-holding alone could account for this difference may be discounted as follows. If it is assumed that all mean intertrial press scores shown in Table 3 up to 80 were due to on-trial disc-holding, then subtraction of 80 from all scores (with scores 80 or below subtracted to zero) should remove the contamination of the artifact. Comparison of the resulting scores by the randomization test for two independent samples reveals that the drug group still has more intertrial presses than the no-drug group ($p=.052$). It seems unlikely that ataxia could have played a role here, for the ataxia noted in drugged Ss during the insoluble problem disappeared within one or two days after the cessation of drug administration. The possibility that unfortunate assignment of monkeys to groups could account

for the difference is ruled out by a comparison of intertrial presses on the last day of pretraining. The mean number of intertrial presses was 131.9 (median, 91) for the no-drug group (n=5) and for the drug group was considerably smaller at 55.0 (median, 48). A two-tailed randomization test for independent samples showed the difference to be not significant, due to large intersubject variation. The possibility of a persisting effect of CDP must be considered since, as was pointed out earlier, squirrel monkeys show behavioral effects one week after a single dosage of CDP. Comparisons of intertrial pressing during the six terminal days of the soluble problem with those during the six post-termination days in a block beginning on the third day after criterion was met revealed significant decreases for two drug-group Ss and suggested that a third drug-group S would also have displayed a significant decrease had she been tested longer; on the other hand, no significant change was shown by the one no-drug S for which the comparison could be made.- Thus, it appears that systemic traces of CDP, or one of its metabolites (Koechlin, Schwartz, Krol, & Oberhansli, 1965), could have contributed to the observed alleviation of shock-suppressed responses. In fact, Randall, Scheckel, and Banziger (1965) reported that the first metabolic product of CDP possessed many of the qualities of CDP when given to mice, rats, and squirrel monkeys. Hence it is

suggested that a persisting effeot of CDP accounted for the elevated intertrial responding of the drug group during the soluble problem.

The 10-14/sec. rhythm observed in EEG records from the amygdala and hippocampus ooourred only when the Ss were motionless and, at least from the posteromedial amygdala, was found to decrease in the presence of CDP.

Lack of movement appeared to be a necessary condition for the appearance of the rhythm. The position of the monkeys when the rhythm occurred was abnormal, for the same Ss were never observed in the home cage to adopt and hold a position with arms held out in space, teeth bared, and eyes closed or half-open and staring blankly. The abnormal posture may represent a species-specific response to noxious stimulation, for the Ss received punishment on a large percentage of trials. Thus the 10-14/sec. rhythm may be a correlate of an inhibition of motor behavior based on fear. Further suggestive evidence that the rhythm was correlated with fear-based motor inhibition is offered by the results of an additional monkey[3] whose amygdala records showed the rhythm to disappear over a period of days without shock and then to reappear when shock was reinstated. The experimenter for this S also noted that rhythmic activity tended to occur only when the

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3. Dr. Robert S. Feldman generously offered the raw data of this S for analysis.

S was motionless. Thus it is suggested that the 10-14/sec. rhythm was correlated with inhibition of motor behavior probably based on fear.

Alternative interpretations of the significance of the rhythm may be offered. One alternative is that the rhythmic EEG activity was an artifact produced by trembling in the Ss when they assumed the abnormal motionless posture. This possibility may be ruled out by the fact that in many Ss the rhythm occurred in only one channel, whereas it would be expected to occur in all channels if it was produced by tremors. A second alternative is that the rhythmic activity represented seizures produced by injury due to the implanted electrodes. Apparent support for this interpretation is offered by the finding of Gergen and MacLean (1961) that the frequency of seizure bursts in the hippocampus of squirrel monkeys was 9-11/sec., which is close to the 10-14/sec. range noted in the present experiment. Arguing against this interpretation, however, are the facts that during seizures the EEG records are flat between bursts of rhythmic activity or contain seizure spikes. In the present study the records showed the interburst intervals to be filled with low voltage fast activity, hence neither flat records nor spikes were observed. Also arguing against this interpretation is the finding of Flynn, Wasman, and Egger (1962) that rhinencephalic seizures disrupt the performance of skeletal re-

sponses while in the present study the Ss would respond immediately upon presentation of a trial despite ongoing rhythmic activity (which ceased as soon as the response was initiated).

Although examination of 29 journals in which EEG studies were likely to be reported from 1960[4] to the present failed to reveal any research on correlative EEGs from squirrel monkeys, or any references to such research, evidence was found which suggested that rhythmic activity from neural centers is associated with inhibitory processes in rats, dogs, cats, monkeys, and humans.

Perhaps the best known of these EEG rhythms is alpha, an 8-13/sec. signal found in humans from scalp electrodes placed over occipital and parietal cortex. Alpha is reduced in amplitude or eliminated by visual stimulation, by attempts to see, or by visual imagery, and hence occurs when the visual system is not in use (Wells, 1962).

A second rhythm, called mu by Glaser (1962, p. 10), is in the 7.5-11/sec. range and is found in records from scalp electrodes on the tempero-parietal areas of humans (Ciganek, 1959) and from deep multielectrodes in the rolandic area of humans (Chatrain, Petersen, & Lazarte,

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4. 1960 was chosen as an earlier limit on the basis of notes, suggesting that squirrel monkeys were rarely used as laboratory animals prior to that time, which appeared in A. M. Sohrier (Ed.), Laboratory Primate Newsletter, Brown University, Providence, R. I.

1959). Authors of the latter two studies reported that passive and reflex movements blocked the rhythm primarily on the contralateral side after the movement was initiated, that spontaneous voluntary movement blocked the rhythm bilaterally with blockade first appearing contralaterally and prior to execution, that voluntary movement on command blocked the rhythm bilaterally, simultaneously, and prior to execution, and that tactile and other stimulation failed to block the rhythm.

Buchwald and co-workers (Buchwald, Wyers, Okuma, & Heuser, 1961; Heuser, Buchwald, & Wyers, 1961; Buchwald, Heuser, Wyers, & Lauprecht, 1961) reported a rhythmic EEG pattern in several motor cortex and subcortical sites which occurred for as long as three sec. in response to a brief electrical pulse to the caudate nucleus of the cat. They called the rhythm the caudate spindle, and found that stimulation intense enought to elicit it decreased the accuracy of visual discrimination performance and drastically reduced speed in a reaction time task. Through use of lesions, drugs, and electrical stimulation these workers identified paths necessary for transmission of the caudate spindle, and hypothesized an interaction between thalamus and caudate such that information from nucleus centrum medianum of the thalamus was fed to the caudate and was fed back to nucleus ventralis anterioris of the thalamus. The information from this "caudate loop" was inhibitory

in nature, and together with facilitatory information ascending directly from nucleus centrum medianum could modify input to the cerebral cortex. Thus the caudate spindle was identified with the action of an inhibitory circuit.

Hippocampograms from rats, rabbits, cats, and monkeys often reveal a 4-7/sec. rhythm called theta by Glaser (1962, p. 10). Green and Arduini (1954) found theta to occur in response to sensory stimulation in any modality in rabbits, cats, and monkeys. Grastyan, Lissak, Madarasz, and Donhoffer (1959) found theta to occur in cats during acquisition of conditioned approach and avoidance responses. Adey, Dunlop, and Hendrix (1960) found theta to occur in cats during approach responses in a discrimination situation. Bremner (1964) found theta to occur in rats in a nondiscriminated avoidance situation. Bremner (1964) suggested that all these results were correlated with an arousal process, possibly underlying attentiveness. Consistent with Bremner's suggestion is the more restrictive view of Gerbrandt (1965) that theta represents an inhibitory process whereby responses irrelevant to or competing with the response to be performed are controlled, thereby allowing release of relevant responses. In other words, in the cases of Green and Arduini (1954) and Grastyan et al. (1959) theta could correlate with an inhibition of attentiveness to background stimuli thereby allowing a focus of attention on the experimental stimula-

tion; in the case of Adey et al. (1960) theta could correlate with the inhibition of the incorrect response tendency in a two-choice situation; and in the case of Bremner (1964) theta could correlate with inhibition of competing responses. Gerbrandt (1964; 1965) uses these and other studies to identify an inhibitory system, "functioning to control behavior competing with a response to be stabilized" (1965, p. 118).

Thus precedents exist in at least four different general areas for rhythmic EEG activity to be associated with inhibitory processes. The 10-14/sec. rhythm observed from the amygdala and hippocampus of squirrel monkeys is perhaps not to be identified with any of the foregoing phenomena due to species, site, and situational differences, but common to all examples is one or another form of inhibition.

Rhythmic activity from the amygdala has been found in several species, but the frequency has typically been very high (40/sec.) compared to that found in squirrel monkeys in the present experiment. The 40/sec. rhythm has been found in cats (John & Killam, 1959), dogs and rhesus monkeys (Domino & Ueki, 1960), and, although related to olfaction in the cat (Carbonell, Escalona, & Sevillano, 1963; Gault & Leaton, 1963), it has been found to serve as an index of arousal (Pagano & Gault, 1964). The latter workers found significant increases in the occurrence of

the rhythm as cats passed from one to the next of the four behavioral states designated "sleep", "relaxed", "alert", and "aroused". Lesse (1960) found that the 40/sec. rhythm from the cat amygdala was specifically elicitable by a CS for an avoidance response, thereby linking the rhythm with emotional arousal. Thus the results of the present experiment, which suggest a relation between rhythmic activity and fear-based motor inhibition, are given some support, at least with regard to the fear aspect, by the findings of Pagano and Gault (1964) and of Lesse (1960).

The effects of CDP on the 10-14/sec. rhythm from the amygdala were found to be a reduction in percentage of time that the rhythm occurred and a reduction in mean duration of bursts. Since, as described earlier, the rhythm appeared to be related to fear, and since CDP not only depresses reactivity of the amygdala to electrical stimulation (Schalleck & Kuehn, 1960; Lanoir, Naquet, & Requin, 1964) but also reduces reactivity of animals to fearful stimuli (Geller, 1962; 1964; Heise & McConnell, 1961) and situations (Feldman, 1962; 1964b; Kamano & Arp, 1962), the reduction in the 10-14/sec. rhythm was perhaps to be expected.

With regard to the appearance of the 10-14/sec. rhythm in hippocampal records, support of a general nature is offered to the notion of an association between rhythmic activity and inhibition of motor behavior by the studies

dealing with the theta rhythm, which showed theta to be associated with selective inhibition of some types of motor behavior. It is unlikely that the rhythm observed in the present experiment may be the squirrel monkey's analog to theta because (1) rhythmic activity in the present experiment was associated with general motor inhibition while theta is correlated with inhibition of a more selective nature, and because (2) the 10-14/sec. rhythm appeared in both amygdala and hippocampus in squirrel monkeys while theta occurs only in the hippocampus of other species. Nonetheless, the general findings of associations between rhythmic neural activity and some types of inhibitory processes in past experiments appear to be consistent with the present findings.

Correlations between the occurrence of the rhythm in the amygdala and in the hippocampus were significantly positive for only one pair of ipsilateral sites. Close examination of the histology suggested that the electrode tips were in the large-celled part of the basolateral amygdala and in the stratum radiatum layer of the posteroventral hippocampus. This pair of sites may be analogous to the pairs in which Kriendler and Steriade (1964a; 1964b) and Morillo (1962a; 1962b) found a strong relationship to exist in terms of evoked potentials. That the remaining correlations between amygdala and hippocampal rhythmic activity were not significant probably reflects non-optimal place-

ment of the tip of one or both electrodes. The remaining hippocampal electrodes from which the rhythm was observed and for which confirmation was possible appeared to have their tips in the pyramidal cell layer.

CDP had a different time course on the 10-14/sec. rhythm from the amygdala than it had on intertrial presses. Despite the increase in rhythmic activity within one to three days noted in Bengt and Ingmar (Table 2, Appendix), intertrial pressing remained elevated for as much as two weeks after removal (Table 1, Appendix). Metabolism of CDP by primates apparently requires a number of days, as behavioral effects of CDP have been observed seven days after a single dosage in cynomolgous monkeys (Geller, 1962) and, as noted before, in squirrel monkeys. It is possible that the 10-14/sec. rhythm is not sensitive to a metabolite of CDP (Koechlin, et al., 1965) which may effect the continued alleviation of response suppression (Randall, et al., 1965). Previous studies of the effects of CDP on the amygdala, such as those of Morillo (1962a; Morillo, Revzin, & Knauss, 1962) and Schalleck and Kuehn (1960), offer no precedents, for spontaneous or correlative activity was not measured. If the intertrial presses and the rhythmic activity are regarded as measures of the same thing, presence of CDP, then the intertrial press is a much more sensitive measure.

However, since ancillary observation suggested that such

gross effects as ataxia, vocalization, and reward acceptance roughly paralleled the time course of recovery of the rhythm after CDP was removed, it is possible that the rhythm and intertrial presses are not measures of the same phenomenon. It is possible that CDP or one of its metabolites could continue to act upon a response-suppression system, as evidenced in the present experiment by continued high rates of pressing between trials, while ceasing to exercise as strong an influence on mechanisms for dealing with fearful stimuli, as evidenced by recovery of or increases in the 10-14/sec. rhythm. Thus it is suggested that CDP allowed separation of two neural systems which may be functionally independent to a degree: one system operates to suppress responses and the other operates to provide appropriate emotional reactions to noxious stimulation. Support for separation of these two systems is offered by Wenzel (1959) who observed that cats given reserpine were still fearful despite alleviation of shock-produced suppression of responses.

If CDP is to be understood as acting principally on the amygdala, then evidence should be available in the lesion and stimulation literature to indicate that the amygdala participates in motor-suppression and fear-appreciation systems. With regard to the motor aspect, the withholding of responses is very difficult for amygdaleotomized rats (Schwartzbaum, Kellicutt, & Thompson, 1964), cats (Horvath,

1963), and monkeys (Schwartzbaum, 1965), and is said by Goddard (1964) in his review to be characteristic of amygdalectomized animals in general. With regard to the emotional aspect, Goddard (1964) cites 43 titles to support the contention that amygdalectomized animals are placid or unresponsive to noxious stimulation, and electrical stimulation of the amygdala has been shown to produce fear responses in rats (Wurtz & Olds, 1963), autonomic arousal in cats (Hilton & Zbrozina, 1963; Lang, Tuovinen, & Valleala, 1964), conditioned avoidance responses in rhesus monkeys (Delgado, Rosvold, & Looney, 1956), and reports of fear from humans (Mullan & Penfield, 1959; Delgado, 1960; King, 1960). None of these studies, however, was conducted with an eye towards separating motor-suppression and fear-appreciation systems, so direct support for the present hypothesis is lacking.

The hypothesis of functionally separable systems within within the amygdala would be aided by reports of topographical separation, but attempts simply to localize functions within the amygdala have met with scant success, probably due to extensive overlap of afferent and efferent projections (Gloor, 1960; Goddard, 1964). Chemical techniques provide a promising means of separating functional systems in spite of overlap. For example, Grossman (1964), by injecting neurochemicals directly into the ventral amygdala of rats, was able to elicit

motivated eating or motivated drinking from a given site, depending on the substance injected. It is doubtful that response suppression could occur without fear, but a demonstration in which fear was observed without suppression would be useful. A demonstration might be based on conditioned emotional response methodology where a three-minute tone terminating with shock is presented while S is responding for food on a variable-interval schedule. The conditioned emotional response is a cessation of responding during tone presentation which develops after a few pairings of tone and shock. If squirrel monkeys with recording electrodes in the amygdala were used, then the 10-14/sec. rhythm might be expected to occur only during tone periods, and emotionality indices such as urination, defecation, vocalization, etc., would be expected to reflect fear during tone periods. Direct injection of CDP into the amygdala would, if the present hypothesis is correct, effect a long-lasting alleviation of suppression during tone periods and a less long-acting depression in the emotionality measures. If the emotionality measures and the rhythmic activity recovered before responses again became suppressed, then support for separable response-suppression and fear-appreciation systems within the amygdala would be offered. Even stronger support would be offered the present interpretation if agents were found which specifically depressed one system and not the other.

In summary, then, CDP tended to produce a decrease in response latencies during the insoluble problem, a long-lasting elevation of punished intertrial responses, and an immediate reduction in the occurrence of a 10-14/sec. EEG rhythm from the amygdala. The latter two effects may be regarded as representing differential action of CDP over time on a response-suppression system and on a fear-appreciation system.

References

1. Adey, W. R., Dunlop, C. W., and Hendrix, C. E. Hippocampal slow waves. Arch. Neurol., 1960, 3, 74-90.

2. Bremner, F. J. The effect of phenelzine on stereotyped behavior in rats. Unpublished M. S. Thesis. University of Massachusetts, Amherst. 1960.

3. Bremner, F. J. Hippocampal activity during avoidance behavior in the rat. J. comp. physiol. Psychol., 1964, 58, 16-22.

4. Buchwald, N. A., Wyers, E. J., Okuma, T., and Heuser, G. The "caudate-spindle" I. Electrophysiological properties. EEG clin. Neurophysiol., 1961, 13, 509-518.

5. Buchwald, N. A., Heuser, G., Wyers, E. J., and Lauprecht, C. W. The "caudate-spindle" III. Inhibition by high frequency stimulation of subcortical centers. EEG clin. Neurophysiol., 1961, 13, 525-530.

6. Carbonell, J., Escalona, J., and Sevillano, M. Modification of the bioelectrical activity of the amygdala after ablation of the olfactory bulbs. EEG clin. Neurophysiol., 1963, 15, 138 (Abstract).

7. Chatrain, G. E., Petersen, M. D., and Lazarte, J. A. The blocking of the rolandic wicket rhythm and some central changes related to movement. EEG clin. Neurophysiol., 1959, 11, 497-510.

8. Ciganek, L. A contribution to the problem of rolandic "arceau" rhythm. EEG clin. Neurophysiol., 1959, 11, 185

(Abstract).

9. Delgado, J. M. R. Emotional behavior in animals and humans. Psychiat. res. Rep., 1960, 12, 259-266.

10. Delgado, J. M. R., Rosvold, H. E., and Looney, E. Evoking conditioned fear by electrical stimulation of subcortical structures in the monkey brain. J. comp. physiol. Psychol., 1956, 49, 373-380.

11. Domino, E. F., and Ueki, S. An analysis of the electrical burst phenomenon in some rhinencephalic structures of the dog and monkey. EEG clin. Neurophysiol., 1960, 12, 635-648.

12. Ellen, P., and Wilson, A. S. Perseveration in the rat followinf hippocampal lesions. Exp. Neurol., 1963, 8, 310-317.

13. Emmers, R., and Akert, K. A Stereotaxic Atlas of the Brain of the Squirrel Monkey (Saimiri sciureus). Madison: University of Wisconsin Press. 1963.

14. Feldman, R. S. The specificity of the fixated response in rats. J. comp. physiol. Psychol., 1953, 46, 487-492.

15. Feldman, R. S. The prevention of fixations with chlordiazepoxide. J. Neuropsychiat., 1962, 3, 254-259.

16. Feldman, R. S. Comparisons of the benzodiazepines with other tranquillizers (sic) during conflict and chronic anxiety states. In P. B. Bradley, F. Flugel, and P. Hoch (Eds.), Neuropsychopharmacology. Vol. 3. Amsterdam:

Elsevier. 1964. (a)

17. Feldman, R. S. Further studies on assay and testing of fixation-preventing psychotropic drugs. Psychopharmacologia, 1964, 6, 130-142.

18. Feldman, R. S., and Green, K. F. Effects of chlordiazepoxide on fixated behavior in squirrel monkeys. J. Psycho-Pharmacol., 1966, 1, 37-45.

19. Feldman, R. S., and Lewis, E. Response differences of psychotropic drugs in rats during chronic anxiety states. J. Neuropsychiat., 1962, 3, S27-S41.

20. Flynn, J. P., Wasman, M., and Egger, M. D. Behavior during propagated hippocampal afterdischarges. In G. H. Glaser (Ed.), EEG and Behavior. New York: Basic Books. 1962. Pp. 134-148.

21. Gault, F. P., and Leaton, R. N. Electrical activity of the olfactory system. EEG clin. Neurophysiol., 1963, 15, 299-304.

22. Geller, I. Use of approach avoidance behavior (conflict) for evaluating depressant drugs. In J. H. Nodine and J. H. Moyer (Eds.), Psychosomatic Medicine. Philadelphia: Lea and Febiger. 1962. Pp. 267-274.

23. Geller, I. Relative potencies of benzodiazepines as measured by their effects on conflict behavior. Arch. int. Pharmacodyn., 1964, 149, 243-247.

24. Gerbrandt, L. K. Generalizations from the distinction of passive and active avoidance. Psychol. Rep., 1964, 15,

11-22.

25. Gerbrandt, L.K. Neural systems of response release and control. Psychol. Bull., 1965, 64, 113-123.

26. Gergen, J. A., and MacLean, P. D. Hippocampal seizures in squirrel monkeys. EEG clin. Neurophysiol., 1961, 13, 316 (Abstract).

27. Gergen, J. A., and MacLean, P. D. A Stereotaxic Atlas of the Squirrel Monkey's Brain (Saimiri sciureus). Bethesda: Public Health Service Publication 933. 1962.

28. Glaser, G. H. The normal electroencephalogram and its reactivity. In G. H. Glaser (Ed), EEG and Behavior. New York: Basic Books. 1962. Pp. 3-23.

29. Gloor, P. Amygdala. J. Field (Ed.), Handbook of Physiology, Vol. 2, Neurophysiology. Washington, D. C.: American Physiological Society, 1960. Pp. 1395-1420.

30. Goddard, G. V. Functions of the amygdala. Psychol. Bull., 1964, 62, 89-109.

31. Grastyan, E., Lissak, K., Madarasz, I., and Donhoffer, H. Hippocampal electrical activity during the development of conditioned reflexes. EEG clin. Neurophysiol., 1959, 11, 409-430.

32. Green, J. D., and Arduini, A. Hippocampal electrical activity in arousal. J. Neurophysiol., 1954, 17, 533-547.

33. Green, K. F., and Moore, J. W. Conditional-outcome choice behavior in squirrel monkeys. Psychon. Sci., 1966, 4, 369-370.

34. Green, K. F., Moore, J. W., and Sargent, T. D. Color preference in squirrel monkeys (Saimiri sciureus). Psychon. Sci., 1966, 4, 367-368.

35. Grossman, S. P. Behavioral effects of chemical stimulation of the ventral amygdala. J. comp. physiol. Psychol., 1964, 57, 29-36.

36. Hanson, H. M. The effects of amitriptylene, imipramine, chlorpromazine, and nialimide on avoidance behavior. Fed. Proc., 1961, 20, 396.

37. Heise, G. H., and McConnell, H. Differences between chlordiazepoxide-type and chlorpromazine-type action in "trace" avoidance. Proc. Third World Congress of Psychiat., 1961, 2, 917-921.

38. Heuser, G., Buchwald, N. A., and Wyers, E. J. The "caudate-spindle" II. Facilitatory and inhibitory caudate-cortical pathways. EEG clin. Neurophysiol., 1961, 13, 519-524.

39. Hilton, S. H., and Zbrozina, A. W. Amygdaloid region for defense reactions and its afferent pathway to the brain stem. J. Physiol., 1963, 165, 160-173.

40. Himwich, H. W. Biochemical and neurophysiological action of psychoactive drugs. In L. Uhr and J. G. Miller (Eds.), Drugs and Behavior. New York: Wiley, 1960. Pp. 41-85.

41. Horvath, F. E. Effects of basolateral amygdalectomy on three types of avoidance behavior in cats. J. comp.

physiol. Psychol., 1963, 56, 380-389.

41. Jarrard, L. E., Isaacson, R. L., and Wicklegren, W. O. Effects of hippocampal ablation and intertrial interval on runway acquisition and extinction. J. comp. physiol. Psychol., 1964, 57, 442-444.

42. John, E. R., and Killam, K. F. Electrophysiological correlates of avoidance conditioning in the cat. J. Pharm. exp. Ther., 1959, 125, 252-274.

43. Kamano, D. K., and Arp, D. J. Effects of chlordiazepoxide (Librium) on the acquisition and extinction of avoidance responses. Psychopharmacologia., 1964, 6, 112-119.

44. Kimble, D. P. The effects of bilateral hippocampal lesions in rats. J. comp. physiol. Psychol., 1963, 56, 273-283.

45. Kimble, D. P., and Kimble, Reeva J., Hippocampectomy and response perseveration in the rat. J. comp. physiol. Psychol., 1965, 60, 474-476.

46. King, H. E. Psychological effects of excitation in the limbic system. In D. E. Sheer (Ed.), Electrical Stimulation of the Brain. Austin: University of Texas Press, 1961. Pp. 477-486.

47. Kluver, H., and Barrera, E., A method for the combined staining of cells and fibers in the nervous system. J. Neuropathol. exp. Neurol., 1953, 12, 400-403.

48. Koechlin, B. A., Scwartz, M. A., Krol, G., and Oberhansli, W. The metabolic fate of C^{14}-labelled chlordiaze-

poxide in man, in tehe dog, and in the rat. J. pharmacol. exp. Ther., 1965, 148, 399-411.

50. Kriendler, A., and Steriade, M. Inhibition of evoked and amygdalo-hippocampal afterdisoharges by administration of eserine. EEG clin. Neurophysiol., 1964, 17, 598 (Abstract). (a)

51. Kriendler, A., and Steriade, M. The effects of allo- and neocortical lesions upon amygdalo-hippocampal paroxysmal afterdischarges. EEG clin. Neurophysiol., 1964, 17, 598 (Abstract). (b)

52. Lang, H., Tuovinen, T., and Valleala, P. Amygdaloid afterdischarges and galvanic skin response. EEG clin. Neurophysiol., 1964, 16, 366-374.

53. Lanoir, J., Naquet, R., and Requin, Comparative experimental study of the anticonvulsant action of chlordiazepoxide (Librium) and diazepam (Valium). Preliminary results. EEG clin. Neurophysiol., 1964, 17, 722-723. (Abstract)

54. Lash, L. Response discriminability and the hippocampus. J. comp. physiol. Psychol., 1964, 57, 251-256.

55. Lesse, H. Rhinencephalic electrophysiological activity during "emotional behavior" in cats. Psychiat. res. Rep., 1960, 12, 224-237.

56. Maier, N. R. F. Frustration: The Study of Behavior Without a Goal. New York: McGraw-Hill, 1949.

57. McGeer, P. Mind, drugs, and behavior. Amer. Scient., 1962, 50, 322-338.

58. Morillo, A. Effects of benzodiazepines upon amygdala and hippocampus of the cat. Int. J. Neuropharmacol., 1962, 1, 353-359. (a)

59. Morillo, A. Participation of the pyriform cortex in the conduction of amygdala hippocampal impulses. Fed. Proc., 1962, 21, 326. (Abstract) (b)

60. Morillo, A., Revzin, A. M., and Knauss, T. Physiological mechanism of action of chlordiazepoxide in cats. Psychopharmacologia, 1962, 3, 386-394.

61. Mullan, S., and Penfield, W. Illusions of comparative interpretation and emotion: Production by epileptic discharge and by electrical stimulation in the temporal cortex. AMA Arch. Neurol. Psychiat., 1959, 81, 269-284.

62. Pagano, R. P., and Gault, F. P. Amygdala activity: A central measure of arousal. EEG clin. Neurophysiol., 1964, 17, 255-260.

63. Peretz, E. Extinction of a food-reinforced response in hippocampectomized cats. J. comp. physiol. Psychol., 1965, 60, 182-185.

64. Randall, L. O., Scheckel, C. L., and Banziger, R. F. Pharmacology of the metabolites of chlordiazepoxide and diazepam. Curr. ther. Res., 1965, 7, 590-606.

65. Schalleck, W., and Kuehn, A. Effects of psychotropic drugs on limbic system of cat. Proc. soc biol. Med., 1960, 105, 114-117.

66. Schalleck, W., Zebransky, F., and Kuehn, A. Effects

of benzodiazepines on central nervous system of cat. Arch. int. Pharmacodyn., 1964, 149, 467-483.

67. Schwartzbaum, J. S. Discrimination behavior after amygdalectomy in monkeys: Visual and somesthetic learning and perceptual capacity. J. comp. physiol. Psychol., 1965, 60, 320-328.

68. Schwartzbaum, J. S., and Poulos, D. A. Discrimination behavior after amygdalectomy in monkeys: Learning set and discrimination reversals. J. comp. physiol. Psychol., 1965, 60, 320-328.

69. Schwartzbaum, J. S., Thompson, J. B., and Kellicutt, M. H. Auditory frequency discrimination and generalization following lesions of the amygdaloid area of rats. J. comp. physiol. Psychol., 1964, 57, 257-266.

70. Sidman, M. Tactics of Scientific Research. New York: Basic Books, 1960.

71. Siegel, S. Nonparametric Statistics for the Behavioral Sciences. New York: McGraw-Hill, 1956.

72. Stollnitz, F. Spatial variables, obsreving responses, and discrimination learning sets. Psychol. Rev., 1965, 72, 247-261.

73. Teitelbaum, H., and Milner, P. Activity changes following partial hippocampal lesions in rats. J. comp. physiol. Psychol., 1963, 56, 987-995.

74. Thompson, R., and Langer, S. K. Deficits in position reversal learning following lesions of the limbic system.

J. comp. physiol. Psychol., 1963, 56, 987-995.

75. Thompson, R., Langer, S. K., and Rich, I. Lesions of the limbic system and short-term memory in albino rats. Brain, 1964, 87, 537-542.

76. Valenstein, E. S., Hodos, W., and Stein, L. A simplified electrode assembly for implanting chronic electrodes in the brain of small animals. Amer. J. Psychol., 1961, 74, 125-128.

77. Wells, C. E. Electroencephalographis correlates of conditioned responses. In G. H. Glaser (Ed.), EEG and Behavior. New York: Basic Books, 1962. Pp. 60-108.

78. Wenzel, B. Relative resistance to reserpine of responses based on positive as compared with negative reinforcement. J. comp. physiol. Psychol., 1959, 52, 673-680.

79. Vernier, V. G., Hanson, H. M., and Stone, C. A. The pharmacodynamics of amitriptylene. In J. H. Nodine and J. H. Moyer (Eds.), Psychosomatic Medicine. Philadelphia: Lea and Febiger, 1962. Pp. 683-690.

80. Wurtz, R. H., and Olds, J. M. Amygdaloid stimulation and operant reinforcement in the rat. J. comp. physiol. Psychol., 1963, 56, 941-949.

Appendix

Table 1. Response data for all Ss on all experimental days until termination criterion was met.

Abbreviations:

GP=Group; ND=No Drug; D=Drug; IP=Insoluble Problem; SP=Soluble Problem; S=Number of on-trial presses to cue opposite the one stereotyped; LAT=Mean Latencies over the four 20-trial blocks of a daily session; +=Correct; -=Incorrect; ITP=Number of Intertrial Presses.

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
ND	Frieda	IP-1	18	2.3*		111	SP-1	0	1.6	1.7	11
		2	0	3.0		80	2	0	2.1	2.2	2
		3	0	2.0		32	3	0	1.4	1.6	25
		4	0	2.0		56	4	0	1.6	1.9	0
		5	0	2.2		11	5	0	2.0	1.7	28
		6	0	1.7		24	6	0	1.0	1.0	11
		7	0	1.4		22	7	0	1.1	0.8	5
		8	0	1.5		63	8	0	0.8	1.0	12

*Latencies during Insoluble Problem are pooled over rewarded and punished trial.

ND	Dodie	IP-1	3	11.4		62	SP-1	0	20.2	19.8	5
		2	9	19.8		3	2	0	19.4	18.5	13
		3	2	20.8		65	3	0	18.2	17.4	3
		4	0	18.9		7	4	0	20.4	19.3	8
		5	0	19.4		9	5	0	20.0	20.0	7
		6	1	21.6		18	6	0	19.9	19.1	8
		7	0	20.5		14	7	0	20.0	19.3	2
		8	0	14.4		53	8	0	20.1	19.3	2
		9	0	14.3		37	9	0	20.1	19.4	7
		10	0	12.8		50	10	0	20.0	20.4	15
		11	1	23.3		4	11	0	20.4	20.5	21
		12	0	21.6		8	12	0	19.0	16.1	8

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
ND	Dodie (Cont.)	IP-13	1	15.9		21	SP-13	0	17.8	18.9	8
		14	0	15.5		5	14	0	20.1	20.3	3
		15	0	16.5		8	15	0	18.1	18.9	7
		16	0	14.9		8	16	0	19.4	19.2	1
		17	0	14.6		12					
		18	0	17.1		39					
		19	0	20.7		5					
ND	Ulrich	IP-1	50	19.4		208	IP-12	1	1.2		104
		2	42	15.6		89	13	0	0.7		14
		3	10	27.7		30	14	0	0.8		0
		4	8	8.8		81	15	0	0.7		4
		5	14	19.8		134	16	0	1.2		5
		6	16	14.4		35	17	0	0.6		17
		7	18	11.7		21	18	0	0.7		2
		8	18	3.5		25					
		9	4	1.2		33	SP-1	0	0.9	0.7	1
		10	2	1.0		28	2	0	0.6	0.6	3
		11	0	0.9		42	3	0	1.0	0.9	14
ND	Anton	IP-1	39	2.0		208	IP-25	0	0.8		104
		2	33	6.4		464	26	0	1.2		106
		3	28	3.5		369	27	1	1.1		91
		4	7	5.1		292	28	2	1.4		58
		5	40	3.3		282	29	0	0.8		86
		6	51	1.6		222	30	4	3.4		12
		7	39	1.8		128	31	0	1.0		83
		8	27	3.0		220	32	0	0.8		56
		9	1	3.0		183	33	0	0.7		21
		10	3	2.0		198	34	0	0.7		24
		11	4	1.8		171	35	0	0.8		23
		12	0	2.1		200	36	0	1.2		18
		13	1	4.2		146					
		14	1	1.4		94	SP-1	0	2.0	0.6	7
		15	0	2.2		97	2	0	0.9	0.6	3
		16	2	1.6		147	3	0	1.1	0.6	10

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
ND	Anton (Cont.)	IP-17	0	3.8		255	SP-4	0	1.1	0.7	15
		18	1	6.2		179	5	0	0.5	1.0	22
		19	1	5.2		217	6	0	0.7	1.6	50
		20	0	2.2		161	7	4	3.0	1.6	70
		21	1	3.2		207	8	1	1.4	1.2	106
		22	2	1.8		257	9	1	0.8	0.7	78
		23	0	1.5		185	10	1	0.7	0.8	79
		24	0	1.2		108	11	0	0.6	0.7	98
ND	Gunnar	SP-1	0	21.8	22.7	185	SP-19	0	14.3	13.6	93
		2	0	21.4	22.4	170	20	0	10.9	11.0	90
		3	0	20.4	21.4	137	21	0	15.6	14.2	79
		4	0	19.7	22.2	159	22	0	15.5	19.4	76
		5	0	19.9	19.7	177	23	0	15.8	17.6	76
		6	0	20.1	20.9	144	24	0	18.5	16.4	140
		7	0	20.4	20.2	157	25	0	16.8	17.0	86
		8	0	23.4	22.7	111	26	0	16.5	16.6	90
		9	0	21.3	21.4	113	27	0	17.3	18.4	83
		10	0	20.7	21.5	72	28	0	18.8	19.4	188
		11	0	20.2	21.2	68	29	0	19.6	19.0	81
		12	0	18.7	16.8	81	30	0	---*---		25
		13	0	16.7	15.6	83	31	0	17.2	19.0	81
		14	0	20.8	21.7	42	32	0	15.4	17.6	64
		15	0	21.6	19.0	69	33	0	13.5	12.0	74
		16	0	14.4	13.4	87	34	0	16.4	14.6	79
		17	0	15.9	14.0	84	35	0	17.8	15.5	82
		18	0	15.5	14.8	82	36	0	18.6	18.6	78

*S refused to respond after Block 2.

D	Phaedra	IP-1	40	25.1		97	SP-1	0	6.6	4.4	10
		2	33	11.7		41	2	0	2.2	2.1	96
		3	4	20.7		54	3	4	5.9	6.7	76
		4	2	7.7		150	4	2	4.9	5.1	56
		5	0	12.9		210	5	4	4.8	6.0	83
		6	0	2.6		117	6	4	2.5	2.9	109
		7	0	2.8		35	7	1	1.1	2.0	93
		8	0	1.8		81	8	2	0.9	1.8	71

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
D	Phaedra (Cont.)	IP-9	0	1.8		51	SP-9	0	0.8	1.1	35
		10	0	2.3		18	10	3	1.6	1.3	58
		11	0	2.3		24	11	2	0.8	0.8	82
		12	0	1.9		15	12	35	1.0		77
							13	48	0.9		75
							14	36	0.9		40
D	Winifred	IP-1	22	20.8		138	IP-17	4	0.6		612
		2	8	26.6		40	18	3	0.6		991
		3	0	23.6		50	19	0	0.9		532
		4	16	2.3		307	20	3	0.8		847
		5	6	1.2		282	21	0	0.6		995
		6	3	1.9		173	22	0	0.5		1089
		7	0	1.5		81	23	0	0.5		1234
		8	4	1.0		359	24	0	0.5		895
		9	0	1.4		27	25	0	0.5		1278
		10	0	1.0		96	26	0	0.6		1104
		11	0	1.0		27					
		12	1	0.9		222	SP-1	1	0.9	0.7	581
		13	0	0.7		376	2	1	2.0	2.1	291
		14	2	0.8		409	3	0	1.1	1.5	215
		15	7	0.9		288	4	0	0.7	0.6	174
		16	10	0.2		743	5	0	0.7	0.6	174
							6	0	0.6	0.6	607
D	Gustav	IP-1	37	41.2		51	SP-1	1	1.3	1.8	76
		2	12	25.7		77	2	0	1.8	1.3	51
		3	12	21.0		88	3	0	1.0	1.1	138
		4	10	2.6		56	4	0	1.4	2.5	30
		5	4	1.9		57	5	0	0.9	0.8	140
		6	0	1.9		41	6	1	1.3	1.1	180
		7	0	2.5		28	7	0	1.0	1.8	21
		8	0	4.6		24	8	0	0.6	0.8	395
		9	0	2.0		10	9	0	0.6	0.7	492
		10	0	1.6		94	10	0	1.0	1.1	64
		11	0	1.1		417					

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
D	Ingmar	IP-1	5	20.2		81	IP-25	0	18.3		1
		2	3	24.5		38	26	0	4.8		12
		3	0	22.2		40	27	0	6.0		64
		4	0	20.4		64					
		5	0	20.8		28	SP-1	2	17.5	16.4	83
		6	3	9.7		142	2	0	10.6	8.3	42
		7	1	13.0		86	3	0	15.3	16.0	8
		8	0	20.8		5	4	0	6.6	7.2	19
		9	0	22.2		0	5	0	2.5	3.8	49
		10	6	18.0		126	6	0	3.0	2.6	23
		11	0	22.8		2	7	0	5.3	6.6	9
		12	0	21.8		5	8	0	4.4	3.0	5
		13	0	21.7		2	9	0	3.5	3.6	5
		14	0	18.3		4	10	0	3.4	4.0	7
		15	1	41.5		0	11	0	4.1	4.2	11
		16	0	20.8		2	12	0	1.8	2.8	8
		17	0	16.8		2	13	0	2.0	1.9	19
		18	0	21.8		1	14	0	3.3	4.3	28
		19	0	22.2		0	15	0	2.1	2.8	3
		20	0	17.6		5	16	0	4.2	3.4	12
		21	1	6.9		83	17	0	2.4	2.5	5
		22	0	16.2		7	18	0	1.8	1.5	4
		23	0	10.8		15	19	0	1.4	1.5	1
		24	0	4.5		24	20	0	1.8	1.2	6
							21	0	1.2	1.4	16
D	Bengt	IP-1	25	28.0		129	IP-33	78	2.8		465
		2	50	23.6		183	34	75	3.0		439
		3	52	21.3		217	35	75	3.2		362
		4	55	24.9		307	36	76	3.4		337
		5	68	22.4		197	37	76	3.0		389
		6	69	20.9		242	38	69	6.3		343
		7	71	16.3		286	39*	77	8.2		474
		8	66	19.5		318	40*	71	9.2		351
		9	66	16.4		432	41*	69	8.2		410
		10	56	12.3		613	42*	72	4.3		469
		11	69	12.6		637	43*	15	21.7		190
		12	73	12.7		585	44*	3	29.2		85

GP	Monkey	Day	S	LAT+	LAT-	ITP	Day	S	LAT+	LAT-	ITP
D	Bengt (Cont.)	IP-13	74	16.0		339	IP-45*	67	29.5		73
		14	79	14.8		383					
		15	72	12.4		583	SP-1	6	14.0	17.9	194
		16	68	12.6		400	2	0	6.6	5.3	323
		17	59	9.8		631	3	0	4.8	3.7	244
		18	65	14.2		441	4	0	2.6	2.0	280
		19	60	16.6		367	5	1	1.8	1.2	256
		20	58	12.0		314	6	0	1.2	1.6	243
		21	58	13.6		418	7	0	1.6	2.5	215
		22	73	11.9		444	8	0	1.3	1.6	199
		23	47	8.8		511	9	0	1.0	1.7	244
		24	63	8.2		401	10	0	1.2	1.8	164
		25	66	4.2		585	11	0	3.2	1.4	115
		26	69	5.2		570	12	0	2.8	3.9	113
		27	75	7.8		399	13	0	2.0	3.7	121
		28	77	7.0		261	14	0	3.7	4.0	112
		29	78	9.5		190	15	0	1.6	2.4	139
		30	71	12.4		127	16	0	1.4	1.5	270
		31	75	4.6		326	17	0	1.0	1.0	159
		32	73	3.6		429	18	0	0.9	0.9	207

*No CDP was given on Days 39-45 in the insoluble problem.

Table 2. Characteristics of the 10-14/sec. rhythm during intertrial intervals from all sites which yielded scorable activity.

Abbreviations:

GP=Group; ND=No Drug; D=Drug; Amyg=Amygdala; Hipp=Hippocampus; L=Left; R=Right; IP=Insoluble Problem; SP=Soluble Problem; %=Percentage of time rhythmic activity occurred; DUR=Mean duration of bursts; T=Total scorable time (sec.); +=After a reward; -=After a punishment.

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Frieda	L.Amyg	IP-1	15	14	1.1	1.0	114	28
			2	18	15	1.0	1.5	168	39
			3	21	24	1.2	1.4	317	125
			4	34	25	1.8	1.8	240	149
			5	20	16	1.3	1.4	262	172
			6	22	11	1.1	1.0	156	70
			7	19	13	1.3	0.7	181	15
			8	17	21	1.3	1.2	237	77
			SP-1	25	20	1.3	1.0	323	156
			2	29	8	1.3	1.0	244	128
			3	13	20	1.2	1.1	156	66
			4	23	9	1.2	0.8	187	86
			5	19	0	1.5	-	80	19
			6	-	-	-	-	0	0
			7	8	20	1.2	1.4	73	66
			8	14	5	1.5	1.2	140	110
		L.Hipp	IP-1	3	4	0.8	1.0	114	28
			2	5	6	0.9	1.0	170	53
			3	5	5	0.8	0.9	317	127
			4	11	7	0.8	1.0	240	149

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Frieda (Cont.)	L.Hipp	IP-5	7	9	0.9	0.9	302	172
			6	20	14	0.8	0.7	128	98
			7	10	7	0.9	0.5	181	15
			8	14	12	0.8	0.8	276	65
			SP-1	16	6	1.0	0.7	333	150
			2	-	-	-	-	0	0
			3	5	3	0.9	1.0	131	39
			4	8	3	0.9	1.5	197	86
			5	3	4	1.0	0.7	88	46
			6	4	4	1.0	1.0	115	51
			7	2	5	0.7	0.8	81	66
			8	8	3	0.7	1.0	140	108
		R.Hipp	IP-1	8	4	0.7	1.0	129	24
			2	6	7	0.8	0.8	170	59
			3	9	10	0.9	0.8	329	105
			4	12	6	1.0	1.0	240	166
			5	6	4	0.9	1.0	262	182
			6	11	17	1.0	1.0	148	63
			7	10	0	0.9	-	181	9
			8	9	9	1.1	0.9	404	209
			SP-1	12	8	1.0	0.8	327	177
			2	8	6	1.0	0.9	244	118
			3	4	10	1.0	1.0	156	63
			4	16	16	0.9	1.0	76	10
			5	18	4	1.1	0.7	85	51
			6	6	6	1.0	1.0	115	51
			7	1	3	1.0	1.0	77	66
			8	4	2	1.2	1.0	140	91
ND	Dodie	R.Amyg	IP-1	21	13	1.1	1.3	336	175
			2	13	24	1.4	1.7	204	119
			3	25	20	1.6	1.2	152	143
			4	17	19	1.4	1.9	235	144
			5	34	31	1.6	1.6	171	90
			6	18	18	1.4	1.6	207	195
			7	21	26	1.3	1.5	224	178
			8	27	24	1.3	1.3	206	165

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Dodie (Cont.)	R.Amyg	IP-9	18	21	1.2	1.3	282	132
			10	6	14	1.0	1.1	208	57
			11	-	-	-	-	0	0
			12	29	17	1.6	1.4	93	66
			13	15	14	1.2	1.2	135	73
			14	-	-	-	-	-	-
			15	21	4	1.5	1.0	87	75
			16	7	12	1.0	1.3	139	67
			17	17	13	1.1	1.0	52	159
			18	-	-	-	-	0	0
			19	-	-	-	-	0	0
			SP-1	-	-	-	-	0	0
			2	-	-	-	-	0	0
			3	-	-	-	-	0	0
			4	-	-	-	-	0	0
			5	-	-	-	-	0	0
			6	-	-	-	-	0	0
			7	-	-	-	-	0	0
			8	18	13	2.0	1.0	45	133
			9	-	-	-	-	0	0
			10	13	7	1.2	1.0	78	81
			11	16	15	1.1	1.8	86	59
			12	17	12	1.1	1.0	117	141
			13	23	13	1.8	0.9	86	102
			14	11	3	1.0	0.4	117	101
			15	14	6	1.2	1.0	98	32
			16	26	9	1.3	0.8	81	78
		L.Hipp	IP-1	6	6	1.0	1.0	336	161
			2	9	17	0.9	1.1	204	119
			3	18	27	0.9	1.0	152	143
			4	13	15	0.9	0.8	225	144
			5	20	13	1.1	1.0	162	94
			6	9	19	0.9	1.0	207	217
			7	15	6	0.9	1.0	224	165
			8	14	5	0.9	1.0	197	167

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Dodie (Cont.)	L.Hipp	IP-9	7	16	0.8	0.9	269	95
			10	12	12	0.8	0.8	185	24
			11	12	18	1.0	0.9	97	50
			12	12	13	0.7	0.9	93	66
			13	14	16	0.8	0.8	116	73
			14	-	-	-	-	-	-
			15	8	11	0.8	0.7	87	70
			16	12	13	0.8	0.9	139	76
			17	8	8	0.8	0.8	74	145
			18	6	7	0.8	0.8	69	71
			19	10	10	0.7	0.9	105	96
			SP-1	14	19	0.9	0.9	128	117
			2	8	10	0.7	0.8	85	150
			3	15	18	0.9	0.8	130	94
			4	14	8	0.9	0.7	93	73
			5	4	15	0.5	1.0	49	127
			6	9	13	0.7	0.8	119	71
			7	19	11	0.9	0.9	135	87
			8	7	18	0.9	0.8	88	112
			9	8	16	0.7	1.0	111	67
			10	10	9	1.0	0.9	78	86
			11	19	18	1.0	1.0	102	39
			12	31	23	1.1	0.9	129	141
			13	12	21	0.8	0.8	86	102
			14	15	22	0.8	0.9	117	101
			15	28	28	1.0	0.9	98	39
			16	17	11	1.2	0.8	65	54
ND	Anton	R.Amyg	IP-1	3	2	1.0	0.8	192	224
			2	0	0	-	-	247	233
			3	0	1	--	1.0	106	256
			4	1	1	1.0	1.0	115	73
			5	1	0	1.0	1.0	139	290
			6	0	0	-	-	103	137
			7	3	2	1.0	1.0	114	262
			8	1	1	1.0	1.0	76	271

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Anton (Cont.)	R.Amyg	IP-9	0	3	-	1.0	99	238
			10	1	2	1.0	0.8	91	250
			11	3	3	1.0	0.7	175	261
			12	0	2	-	0.8	145	275
			13	4	2	0.9	0.9	217	413
			14	1	2	1.0	0.9	148	283
			15	2	1	1.0	0.7	151	181
			16	7	6	1.1	1.0	130	301
			17	2	1	0.7	1.0	177	134
			18	4	3	1.0	0.9	104	204
			19	2	1	0.7	1.0	294	229
			20	0	2	-	1.0	53	200
			21	8	3	1.0	0.8	48	178
			22	4	3	0.7	1.0	207	144
			23	1	2	0.5	0.8	166	140
			24	5	3	1.5	1.3	57	60
			25	3	4	0.8	0.8	154	204
			26	3	2	1.0	0.8	119	431
			27	2	3	1.0	0.9	110	448
			28	4	5	1.0	1.0	130	236
			29	2	1	1.0	0.5	98	161
			30	6	3	0.8	0.9	252	185
			31	4	5	0.8	1.0	156	154
			32	2	2	1.0	0.8	111	234
			33	4	1	1.0	1.0	75	267
			34	2	3	1.5	1.3	197	156
			35	6	0	1.0	-	188	135
			36	4	2	1.0	0.8	137	276
			SP-1	0	0	1,0	1.0	449	453
			2	1	0	1.0	-	155	173
			3	5	2	0.9	1.0	183	169
			4	0	0	-	-	149	134
			5	3	3	0.8	0.9	327	232
			6	1	2	0.7	1.0	135	143
			7	6	1	0.9	1.0	201	338
			8	4	5	1.0	0.8	145	133
			9	1	1	1.0	1.0	208	140
			10	2	4	0.8	0.9	197	152
			11	2	1	0.6	1.0	192	132

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
ND	Gunnar	R.Amyg	SP-1	6	5	0.8	0.9	159	154
			2	3	2	0.7	1.0	166	137
			3	1	3	0.7	0.8	173	165
			4	3	4	0.6	1.0	186	180
			5	2	1	0.8	1.0	145	102
			6	3	4	0.7	1.2	186	142
			7	2	2	1.0	0.8	115	126
			8	4	3	0.8	1.0	214	150
			9	3	3	0.7	0.7	203	229
			10	5	3	0.8	0.7	204	188
			11	2	1	0.8	1.0	258	118
			12	7	6	0.9	1.1	177	200
			13	8	7	0.8	1.0	197	136
			14	3	2	0.8	0.8	183	129
			15	13	4	1.0	0.8	176	151
			16	4	4	0.7	0.8	199	161
			17	4	8	0.9	0.7	166	172
			18	9	6	1.1	0.7	171	142
			19	8	6	1.0	0.9	120	194
			20	2	1	1.0	0.5	211	166
			21	2	2	1.0	0.8	157	147
			22	4	4	0.8	0.8	230	142
			23	4	6	1.0	1.2	244	121
			24	1	2	1.0	0.7	105	217
			25	1	1	1.0	1.0	165	145
			26	0	0	-	-	196	154
			27	1	1	1.0	0.7	158	151
			28	3	1	0.7	1.0	219	155
			29	3	1	0.8	1.0	205	153
			30	2	2	1.0	0.5	101	127
			31	1	4	1.0	1.0	182	160
			32	0	0	-	-	206	155
			33	1	1	1.0	1.0	159	126
			34	1	1	1.0	1.0	181	116
			35	1	0	1.0	-	158	191
			36	0	0	-	-	166	183

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Phaedra	L.Amyg	IP-1	-	-	-	-	-	-
			2	45	38	2.0	2.0	188	225
			3	19	17	1.6	1.4	127	156
			4	41	37	2.3	1.5	196	184
			5	21	30	2.1	1.4	143	156
			6	27	33	1.4	1.3	157	119
			7	26	42	1.5	1.5	252	163
			8	31	31	1.8	1.9	203	183
			9	29	23	1.8	1.8	447	125
			10	18	21	1.7	1.7	241	94
			11	39	26	1.8	1.7	111	83
			12	20	17	1.5	1.4	123	77
			SP-1	17	26	1.4	2.2	106	34
			2	24	8	1.3	1.5	80	47
			3	26	3	1.8	0.7	157	72
			4	31	32	2.2	1.7	163	60
			5	49	41	2.3	2.1	89	93
			6	33	29	2.4	3.0	116	73
			7	39	31	3.6	2.5	64	86
			8	31	0	2.0	-	65	43
			9	28	16	2.1	3.5	91	45
			10	30	17	2.2	1.3	86	77
			11	-	-	-	-	-	-
			12	25	21	2.1	1.9	114	82
			13	-	-	-	-	-	-
			14	55	44	5.8	2.4	211	137
		R.Amyg	IP-1	-	-	-	-	-	-
			2	57	50	3.4	2.8	188	226
			3	29	31	2.3	1.4	127	156
			4	53	61	3.0	2.2	196	184
			5	20	46	1.6	1.9	137	162
			6	45	55	2.1	2.0	157	119
			7	42	55	3.2	2.2	252	163
			8	37	36	2.7	1.7	203	183
			9	37	38	2.6	2.3	483	109
			10	22	27	2.0	2.1	241	94
			11	46	53	2.8	2.2	111	83
			12	27	31	1.6	1.2	123	77

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Phaedra (Cont.)	R.Amyg.	SP-1	25	56	2.7	2.1	106	34
			2	34	28	3.4	2.6	80	47
			3	35	15	2.6	1.8	157	72
			4	42	42	2.5	3.6	163	60
			5	62	59	4.0	2.9	84	83
			6	-	-	-	-	-	-
			7	-	-	-	-	-	-
			8	25	26	1.9	3.3	52	38
			9	-	-	-	-	-	-
			10	-	-	-	-	-	-
			11	33	48	2.3	2.8	144	52
			12	-	-	-	-	-	-
			13	31	30	3.1	2.4	140	57
		L.Hipp	IP-1	-	-	-	-	-	-
			2	30	30	1.5	1.6	191	225
			3	14	19	1.1	1.1	113	156
			4	24	22	1.3	1.2	229	195
			5	15	17	1.3	0.9	143	171
			6	19	11	1.1	1.0	132	144
			7	16	23	1.3	1.1	237	177
			8	23	17	1.7	1.1	203	183
			9	15	11	1.4	1.5	434	178
			10	5	9	1.2	1.1	214	94
			11	15	18	1.0	1.7	48	28
			12	7	5	1.3	0.8	109	77
			SP-1	7	12	0.8	1.0	106	42
			2	14	5	1.4	1.5	76	66
			3	15	4	1.2	1.0	157	54
			4	19	10	1.1	0.9	151	60
			5	27	20	1.5	1.2	89	93
			6	11	12	1.0	1.2	53	49
			7	18	6	1.2	1.0	79	66
			8	16	9	1.1	1.2	117	81
			9	16	10	1.2	1.2	91	50
			10	23	10	1.5	0.7	86	77
			11	9	6	1.0	1.0	125	52
			12	10	18	0.9	1.1	139	57

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Phaedra (Cont.)	L.Hipp	SP-13	16	14	1.6	1.3	140	57
			14	22	24	1.3	1.0	200	137
		R. Hipp	IP-1	-	-	-	-	-	-
			2	36	33	1.5	1.8	191	225
			3	17	19	1.1	1.0	127	156
			4	25	18	1.6	1.3	229	206
			5	19	23	1.0	1.5	143	171
			6	15	13	1.4	1.2	157	117
			7	20	28	1.5	1.4	252	163
			8	26	21	1.9	1.5	203	183
			9	26	18	1.6	1.3	348	132
			10	12	11	1.3	1.1	241	94
			11	20	18	1.5	1.2	111	83
			12	14	12	1.0	1.1	72	66
			SP-1	10	26	1.4	1.1	101	42
			2	14	8	0.9	1.0	83	60
			3	22	5	1.6	1.2	151	100
			4	17	13	1.5	1.1	163	60
			5	39	34	1.4	1.4	84	93
			6	27	26	1.6	1.2	116	73
			7	26	18	1.4	1.2	53	92
			8	23	7	1.8	1.2	117	81
			9	19	27	1.3	1.3	91	45
			10	27	23	1.6	1.3	86	77
			11	15	29	1.5	1.2	144	52
			12	23	21	1.3	1.4	115	82
			13	17	23	1.5	1.9	136	57
			14	33	42	1.8	1.8	211	141
D	Ingmar	R.Amyg	IP-1	24	16	1.4	1.1	215	348
			2	11	19	1.1	1.2	169	249
			3	19	21	1.4	1.3	297	177
			4	27	18	1.4	1.3	148	242

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Ingmar (Cont.)	R.Amyg	IP-5	11	13	0.9	1.5	218	90
			6	10	10	1.8	1.3	164	141
			7	22	21	1.1	2.0	222	94
			8	16	24	1.1	1.1	110	128
			9	23	15	1.4	1.0	144	193
			10	0	2	-	1.0	195	111
			11	16	10	1.6	1.2	215	192
			12	16	17	1.3	1.1	238	154
			13	12	13	0.9	1.0	137	428
			14	13	12	1.0	1.2	222	112
			15	16	19	1.3	1.3	273	134
			16	23	5	1.2	0.9	342	163
			17	14	10	1.0	1.0	150	249
			18	14	30	1.2	1.0	43	92
			19	22	23	1.2	1.2	178	312
			20	13	29	0.9	1.2	179	191
			21	5	1	1.2	1.0	203	179
			22	7	4	0.9	1.0	164	146
			23	3	5	0.8	1.0	145	256
			24	20	18	1.2	1.0	153	132
			25	19	13	1.5	1.3	342	110
			26	14	21	1.1	0.9	167	160
			27	16	17	1.2	1.0	239	155
			SP-1	27	34	2.0	1.9	188	143
			2	21	23	1.8	1.6	265	175
			3	33	34	1.9	1.6	243	204
			4	33	24	1.6	1.4	209	139
			5	35	43	1.7	1.8	231	192
			6	33	36	1.8	1.3	178	155
			7	33	33	1.7	1.5	251	184
			8	37	43	2.0	2.3	160	174
			9	31	34	2.1	2.0	247	233
			10	42	42	2.0	1.9	246	184
			11	36	37	2.4	2.1	195	140
			12	32	32	1.9	1.8	299	170
			13	38	33	2.0	2.0	208	219
			14	28	28	2.0	2.6	222	194
			15	31	34	1.7	1.2	227	157
			16	24	33	1.6	2.3	166	166

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Ingmar (Cont.)	R.Amyg	SP-17	30	31	1.7	1.7	381	294
			18	32	22	2.0	1.7	174	162
			19	24	35	1.3	1.8	179	173
			20	22	21	1.9	1.6	182	218
			21	32	14	1.7	1.6	199	168
D	Bengt	R.Amyg	IP-1	2	4	1.0	0.9	172	206
			2	2	2	0.5	1.0	40	215
			3	3	1	0.8	1.0	97	243
			4	3	2	1.0	0.8	114	264
			5	1	2	1.0	0.7	142	245
			6	0	0	-	-	95	196
			7	0	0	-	1.0	54	316
			8	2	0	1.0	1.0	119	214
			9	0	1	-	1.0	76	198
			10	0	0	-	-	67	170
			11	0	0	-	-	90	262
			12	0	0	-	-	97	163
			13	0	0	-	-	132	197
			14	0	0	-	-	87	249
			15	0	0	-	-	101	184
			16	0	0	-	-	63	299
			17	0	0	-	-	103	125
			18	0	0	-	1.0	64	325
			19	0	0	-	-	146	269
			20	0	0	-	-	109	115
			21	0	0	-	-	24	217
			22	0	0	-	-	96	256
			23	0	0	-	-	60	191
			24	-	-	-	-	0	0
			25	0	0	-	-	5	116
			26	0	0	-	-	73	58
			27	0	0	-	-	93	200
			28	0	0	-	-	98	171
			29	2	0	1.0	0.5	131	292
			30	0	2	-	1.0	118	131
			31	1	0	1.0	-	60	294
			32	1	1	1.0	1.0	107	187

GP	Monkey	Site	Day	%+	%-	DUR+	DUR-	T+	T-
D	Bengt (Cont.)	R.Amyg	IP-33	0	1	-	1.0	101	173
			34	0	1	-	0.5	48	182
			35	1	1	1.0	1.0	61	178
			36	0	0	-	1.0	90	259
			37	1	1	1.0	1.0	104	249
			38	0	0	-	-	128	169
			39*	0	0	-	-	40	73
			40*	6	8	1.2	0.9	119	228
			41*	12	17	1.5	1.5	100	185
			42*	0	6	-	1.3	67	202
			43*	10	9	1.0	1.1	153	212
			44*	7	3	0.8	0.7	181	223
			45*	12	8	1.2	0.9	167	225
			SP-1	7	3	0.9	1.0	121	156
			2	12	4	1.2	0.8	110	107
			3	21	21	1.0	1.1	150	104
			4	16	19	0.9	1.0	140	110
			5	11	9	1.0	0.9	147	136
			6	6	9	1.0	0.8	119	104
			7	9	6	0.9	1.0	164	96
			8	2	9	1.0	0.9	128	140
			9	17	15	1.0	1.1	106	117
			10	7	3	0.9	1.0	190	109
			11	11	10	0.9	1.1	134	135
			12	9	10	1.2	1.0	156	224
			13	12	11	1.1	1.2	213	126
			14	12	18	1.3	1.2	137	143
			15	2	6	1.0	1.2	113	123
			16	0	0	-	-	95	90
			17	10	10	1.1	1.0	121	112
			18	6	10	1.2	1.1	119	147

*No CDP was given on Days 39-45 of the insoluble problem.